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RECRUITMENT TESTS AT LOW SENSATION LEVELS.*

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Testing the loudness recruitment phenomenon is invaluable in routine clinical work. It can materially assist the otologist in arriving at a more definite diagnosis as to the site of pathology than was possible less than ten years ago.¹ In addition to the diagnostic point of view, recruiting ears form a separate class in rehabilitation, because the functional properties of these ears often differ fundamentally from those of ears without recruitment.

Although the recruitment tests proper—Fowler's binaural and Reger's monaural balance—can be performed in a great majority of patients,² there remain a few cases where the deafness in both ears and at all frequencies is so similar that neither of the two procedures can be employed satisfactorily. It is in this comparatively rare connection that the need for a good substitute for the balance test arises.

Certain special features of some recruiting ears are frequently elaborated upon and presented as being characteristic of the whole group; it is regrettable, however, that most of the original studies have not been fully validated against the classical loudness balance tests and have proved disappointing in critical control experiments.

One of the characteristics thought by some investigators to

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label all cases of recruiting deafness, is a less than normal intensity difference limen (IDL). The inconsistencies of this reasoning were sufficiently stressed earlier² and there is no need to repeat here the arguments against any close relation between recruitment and various IDL tests. Control experiments have shown unquestionably that at least for the present there are no such IDL tests as might be substituted for the proper loudness balance measurement.^{2,3,4,5}

Hallpike and Hood, and their group have repeatedly stressed the occurrence of another characteristic of recruiting ears; *viz.*, the development of pathological per-stimulatory adaptation (also called per-stimulatory fatigue.^{6,7,8,9} In their experiences a recruiting ear cannot sustain the initially perceived loudness (at sufficiently high intensity levels) and fails in performance. During continuous three-minute stimulations a clear decrease in loudness follows; the initial difference at threshold may again appear also at louder levels between the ears, or the decrease may even result in the reversal of loudness recruitment. In other words, the ear fails so much that the initial threshold difference increases at high sensation levels.

Dix and Hood,⁷ and Hood,⁹ have carried the weight of these findings into clinical application. Because, as they think, a small IDL is a corollary phenomenon with loudness recruitment, the size of the IDL must also follow the fluctuations perceived in the loudness of sustained tones in a recruiting ear. This was exemplified with threshold tracings in two cases of Ménière's disease; automatic recording showing excursions of the width of less than 5 db during the first minute, while after two or three minutes' stimulation they increased to as much as 15 db. In addition, the pathological adaptation manifested itself by a decrease in sensitivity; after three minutes' testing the threshold was about 15 db worse than at the beginning of the test.

On the basis of these findings Dix and Hood⁷ doubted the validity of the results of those authors who had earlier opposed the use of IDL tests. The main argument against Lund-Iwersen⁴ and against Liden and Nilsson³ was that they used

sustained tones while measuring recruitment or IDL, and, therefore, their results could not be considered reliable.

It is quite true that to demonstrate loudness recruitment, the proper procedure is to use interrupted stimuli alternately in the ears, preferably with a short interval between the pair of tones. The point of the two articles, however, was not so much in recruitment testing but in the fact that the size of the IDL was very much the same in all different groups of deafness, or in normals. Had the test results of these studies been faulty, as Dix and Hood⁷ imply, then they would have been manifested in larger than normal IDLs for recruiting cases owing to sustained stimulation and pathological adaptation; however, this was not the case.

In a discussion of Hood's⁸ paper in 1953, I¹⁰ pointed out some of the inconsistencies in the assumed close relation between a small IDL and loudness recruitment on theoretical grounds, and by reviewing some of the contradictory studies published on the subject. It was concluded that while a small IDL may often be true in recruitment, it does not necessarily mean that it would be otherwise in any other group of deafness, and that a fairly large IDL can also appear in recruiting cases.

It was also shown later¹¹ that the occurrence of pathological adaptation is not so common a phenomenon in recruiting deafness as to jeopardize all tests that have been made with sustained tones. In a series of 51 recruiting ears only 14 per cent showed as large decreases at the loudness levels as would be needed to abolish the recruitment phenomenon and to differentiate them from normal ears.

While dealing with pathological adaptation at threshold level, Dix and Hood⁷ stated that diminished sensitivity during sustained stimulation occurs also in a variety of other perceptive disorders, including lesions of the VIIIth nerve. Although they considered the physiological basis of this phenomenon to be different in these cases, they concluded that a loss in the threshold sensitivity is by no means an indication of the presence or absence of loudness recruitment.

In a later paper Hood¹² regarded this test as valuable in the clinical differentiation of end-organ disease. The amount of sensitivity loss during a modified threshold stimulation was from 20 to 30 db during only 70 seconds stimulation. The possible occurrence of this phenomenon in nonrecruiting perceptive deafness was not dealt with.

Decreased sensitivity during continuous threshold stimulation has been noted also by other investigators. It was reported in three patients by Reger and Kos¹³ in 1952: the amount of the shift varied from 10 to 30 db. Two of the cases were recruiting, the third a verified case of acoustic tumor. In the latter case the sensitivity loss was most marked.

These observers pointed out, however, that all individuals with recruitment did not show temporary threshold shift, nor did it occur in every case of nonrecruiting perceptive deafness. Recently Kos¹⁴ again reported similar threshold shifts in a case of VIIIth nerve atrophy and in a case of pinealoma, confirmed by surgery.

In their latest papers Hood¹² and Kos¹⁴ seem to hold slightly diverging opinions regarding the occurrence of threshold sensitivity loss. Both have earlier expressed their view of this phenomenon occurring in both end-organ and nerve fiber deafness.^{7,13} Hood now speaks of it as one characteristic of end-organ deafness while Kos supports mainly the opposite view.

Dix and Hood,⁷ Hood,⁹ and Reger and Kos¹³ have all implied that a small IDL, diminished excursions in the threshold audiogram and recruitment are equivalent. In fact the two first mentioned phenomena are considered to be measures of the IDL, and this term is used interchangeably for either measure. It is probable that small threshold variability and recruitment go parallel, but not, as stated earlier,^{2,15} because a Békésy-type audiogram is a measure of a true IDL but rather because it is a measure of the variability around the patient's threshold. Small variability indicates that the audibility goes up rapidly; large variability is an indication of a clearly slower relation between the two. It must be stressed here again that it fur-

nishes us with the knowledge of this relation only at the threshold level; in fact, according to the observations of Harris, Haines and Myers¹⁶ on various types of recruitment function, one is quite uncertain when evaluating the presence or absence of recruitment from the Békésy-type curve alone. True, one might not be much misguided in cases where the threshold excursions are small, but might fail in many cases with normal excursions but complete recruitment at louder levels.

Earlier, when measuring pathological adaptation at above threshold levels, the writer¹¹ also made some preliminary records of sustained tone threshold recordings during four minutes' stimulation. In this paper I supplemented these findings on the basis of a greater number of cases. An attempt is also made to study the relation between the recruitment phenomenon and the width of the excursions measured with a self-recording audiometer to reveal the possible advantages and limitations of the latter method in expressing the presence of recruitment.

TESTING PROCEDURE AND APPARATUS.

Air and bone conduction thresholds were measured at frequencies one or one-half octaves apart, employing a Peters SPD-2 audiometer. The threshold was determined with the method of limits using interrupted, about 1 to 2 seconds stimuli. The PDR-10 earphones had reasonably flat response characteristics.

Recruitment testing was done by applying tones of 1.5 seconds duration with an interval of 0.5 seconds alternately to the ears. Either Fowler's binaural or Reger's monaural technique was used. A frequency was selected at which the other ear was normal, or in Reger's balance, a neighboring frequency in the same ear was normal.

If both ears showed decreased sensitivity but in unequal quantities, the balancing procedure was nevertheless employed. The smallest acceptable difference between the ears, or frequencies, was 20 db, the highest 55 db. These differences

apply also in cases where the other ear was normal; with this method the danger of shadow hearing was eliminated.

In both procedures a balance was obtained at settings of 20, 40, 60, 80 and 100 db above the threshold of the better ear. The highest intensity obtainable was 100 db above zero, corresponding to 116 db re 0.0002 microbar.

At each balancing level at least three different judgments of equal loudness were obtained. The equality was approached both from "above" and "below,"² and the patient was allowed

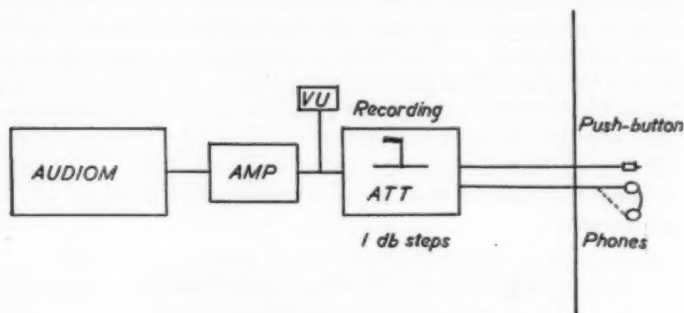


Fig. 1. Block diagram of apparatus.

to listen to as many pairs of tones as he wished before giving the judgment of "equal" or "different." A case was labeled recruiting only if the difference at threshold disappeared completely at louder levels.

The threshold audiometry using sustained tones was done with the method of adjustment. The Peters SPD-2 audiometer was used as a separate sound source to feed the tones to the first channel of the Békésy-type audiometer. The intensity of the tones was controlled before the attenuators with a Pegel voltmeter. The block diagram of the apparatus is shown in Fig. 1.

The listener controlled the intensity of the tones in his ear by means of a push-button which regulated the intensity in one db steps at a rate of 140 db per minute and operated the

instantly reversible motor moving the audiogram chart. The subject was instructed to push the button every time he heard a tone, and release it as soon as the tones disappeared. The recording pen traced the excursions automatically in the adjacent room.

The width of the excursions demonstrates directly the amount of variability around the threshold as a function of time. The changes in sensitivity are manifested by either slowly ascending or descending threshold curves, and if there is no change, the tracing pursues a steady course.

In certain cases supplementary speech tests were made. The technique followed the principles described earlier.¹⁷

Finally, when it was necessary, thermal noise was delivered through the second channel of the Békésy-type audiometer for masking purposes. The principles involved and exact intensities used are adequately described elsewhere.¹⁸

MATERIAL.

The great majority of the persons on whose records this report is based are patients in my private practice. They represent all the various groups of deafness, although attention is focused mainly on patients with perceptive deafness. Unfortunately, cases with nonrecruiting perceptive deafness are few in number, and the material includes no patients with acoustic tumors; on the other hand, recruiting perceptive deafness is sufficiently represented.

Much attention is given to good patient-tester relationship in the Audiology Laboratory. If the subject does not fully understand his share in the test, there is little hope for accuracy in the results. The special tests were explained as simply and comprehensively as possible.

The familiarity of the patient with auditory testing may give him an advantage over a person undergoing both the ordinary threshold tests and supplementary special tests for the first time. This point was kept in mind while analyzing the results.

The conductive group consisted of six persons. Two were under treatment for subchronic otitis media, two were unilateral cases of adhesive otitis, and two were patients with otosclerosis of rather short duration; their hearing loss being still mainly unilateral. All showed good bone conduction and normal intelligibility for speech.

The nonrecruiting perceptive group includes two persons with presbycusis and with a prominent high tone loss: one of them (O. E.) also had a superimposed conductive lesion. In four cases of partial VIIIth nerve degeneration the etiology could not be clearly defined.

In the recruiting perceptive group 27 cases were studied. The diagnosis was Ménière's disease in 10 cases, noise trauma in two cases, and in 15 cases the etiology could not be definitely determined.

RESULTS.

If a person with normal hearing makes a threshold tracing with a self-recording apparatus, the tracing pursues a steady course indefinitely;¹³ however, small fluctuations in this course (of the order of ± 5 db) should be considered normal. This amount of change can easily be caused by slight inattention while listening, while shifting position or breathing deeply, or it may arise if the noise level of the room undergoes small variations. Using the method of limits, ± 5 db is also generally considered to represent the experimental error.

All the individual results are presented in Table I. The threshold at the tested frequency, the amount of temporary threshold shift after two, three and four minutes of sustained stimulation, the width of the excursions during the first minute of recording, the amount of change in the amplitude of the excursions during stimulation, and finally the diagnosis, appear in the table.

The reference intensity for the temporary threshold shift is the midpoint of the excursions obtained during the first half minute of stimulation. In some cases, in which the initial

trend was not clear, the midpoint of the tracings obtained during the first minute of stimulation was used.

In the conductive cases the threshold tracings lay on both sides of the reference level, and none of the values deviated from it by more than 5 db. It seems that the threshold remained stable, and the ears did not become less sensitive during sustained stimulation.

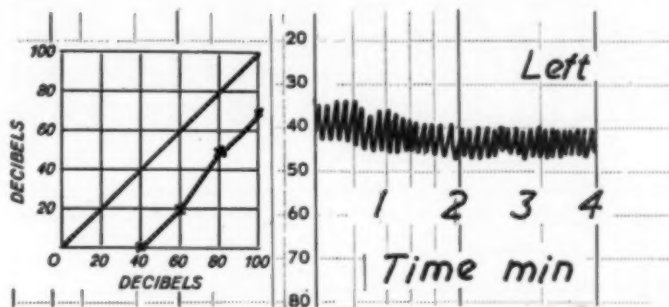


Fig. 2. Case A. M., pharmacist, age 37 years. The patient has adhesive otitis in the left ear. The audiograms showed normal hearing in the right ear and a moderate 20-40 db conductive hearing loss in the left ear.

The binaural loudness balance test at 500 cps. shows no recruitment. The four minutes threshold tracing at the same frequency shows a normal course with some reduction in the width of the excursions during the last two minutes.

The width of the excursions during the first minute of stimulation varied somewhat from individual to individual. It seems fair to say that generally they averaged 7 to 8 db, the lower limit being around 5 db and the upper below 15.

The width of the excursions did not change much during stimulation. The change was of the order of $\pm 1-2$ db, except in one case with the largest initial variability (A. M.). Here the final tracing was 7 to 8 db; there was not so much change in the lower limit of the tracing, but the upper limit (the just-not-heard-tone) decreased relatively much more.

All these patients had had audiometric tests made earlier. Fig. 2 shows the results in one case (A. M.).

In the nonrecruiting perceptive group the initial width of

TABLE I.
INDIVIDUAL RESULTS.

Case	Threshold db	Frequency cps.	Threshold		Initial Width db	Excursions		Diagnosis
			2 Min.	3 Min.		Change During Stimulation db		
H. L.	40	500	0	-1	7-8	Larger by 1 db	Conductive deafness; no recruitment	
S. L.	30	1000 dx	+3	+4	6-8	Larger by 1-2 db		
T. S.	50	1000 sin	-2	-2	7-8	No change		
	10	1000 dx	-1	-1	5-7	No change		
A. M.	45	1000 sin	-2	+4	5-6	Larger by 1-2 db	Conductive deafness; no recruitment	
A. A.	40	500	+4	+4	8-12	Smaller by 3-5 db		
A. A.	30	1000	+2	0	7-8	Smaller by 1-2 db		
T. P.	20	1000	+2	0	6-10	No change		
T. H.	40	1000 sin	-3	-4	7-8	Smaller by 23 db	Perceptive deafness; no recruitment	
	60	1000 dx	-2	-4	7-8	Smaller by 23 db		
M. L.	15	1000 sin	-1	-1	6-8	No change		
	40	1000 dx	-1	+3	6-8	Smaller by 1-3 db		
E. L.	40	6000 sin	-2	+4	6-8	Larger by 23 db	Perceptive deafness; no recruitment	
	50	6000 dx	+2	-2	10-15	Larger by 23 db		
A. W.	20	250	-1	-2	10-16	Smaller by 3-4 db		
O. E.	50	2000	+2	+5	7-9	Smaller by 2-3 db		
K. K.	15	2000	-1	+1	3-4	No change	Perceptive deafness with asymptotic recruitment	
	40	4000	+1	+1	3-4	No change		
U. M.	35	4000 sin	+9	+20	1-3	Smaller by 1 db		
	65	4000 dx	+7	+7	1-2	No change		
M. M.	45	1500 sin	+3	+4	2-3	No change	Perceptive deafness with asymptotic recruitment	
	70	1500 dx	+3	+2	2-3	No change		
T. S.	20	250 dx	+4	+6	7-10	Smaller by 1-3 db		
	40	250 sin	+1	+1	6-8	Larger by 1-2 db		
P. L.	75	3000	0	0	3-5	No change	Perceptive deafness with asymptotic recruitment	
M. V.	40	6000	+3	+5	2-3	Smaller by 1 db		
A. P.	30	4000	-4	-2	3-4	Smaller by 1-3 db		

TABLE I—Continued.

Case	Threshold db	Frequency cps.	Threshold Shift db			Initial Width db	Excursions Change During Stimulation db	Diagnosis
			2 Min.	3 Min.	4 Min.			
V. K.	75	500	+5	+5	+4	10-11	No change	Perceptive deafness with asymptotic recruitment
	70	1000	+2	+3	+3	5-7	Smaller by 3 db	
	55	2000	+7	+6	+6	5-7	No change	
	60	4000 dx	+2	+4	+3	5-8	Smaller by 4 db	
V. U.	50	4000 sin	+2	+5	+3	8-10	Smaller by 4-5 db	
	30	4000 sin	+2	+3	+2	3-5	No change	
	15	2000 sin	0	+1	+2	7-10	No change	
	65	1000	+4	+4	+4	3-5	No change	
S. A.	30	4000	-2	+1	+2	4-5	No change	
T. L.	45	4000	-1	+1	0	2-3	No change	
E. U.	45	4000	-1	+3	-4	2-3	No change	
P. L.	45	4000	+4	+4	+6	4-7	No change	
U. A.	70	4000	+4	+4	+4	4-7	No change	
U. L.	30	500	+1	-1	-1	7-10	No change	Perceptive deafness, with straight line recruitment
	60	1000	+2	+1	-3	8-10	No change	
	20	4000 dx	-3	-6	-5	7-10	Smaller by 4-6 db	
	50	4000 sin	-2	-4	-3	2-3	No change	
	45	250	-1	+3	+3	6-9	No change	
	35	500	-1	+1	+1	7-8	No change	
	65	3000	0	+1	+1	9-12	No change	
	60	1000	0	+0	+5	10-12	Larger by 4-6 db	
	30	500	-1	-1	-1	5-7	No change	
	50	1000	-4	-5	-5	8-10	No change	
V. U.	25	250	-2	+1	+2	8-9	No change	Perceptive deafness, with delayed recruitment
K. K.	50	500	0	+2	+4	5-8	No change	
V. V.	30	500	+3	+2	+2	13-16	No change	
K. V.	30	500	-2	-4	-5	18-20	No change	
	45	1000	+3	+3	+2	15-18	No change	
	35	2000	+5	+6	+8	15-16	No change	
	45	4000	+3	+6	+8	10-16	No change	

the excursions was about the same as in the conductive group, viz., on an average 7 to 8 db. In this group, too, the case showing largest variability (A. W.) at the beginning, exhibited a clear decrease during the test. Except in one case (E. L.), which is shown in Fig. 3, the amplitude of the tracings generally decreased by a few decibels during sustained stimulation.

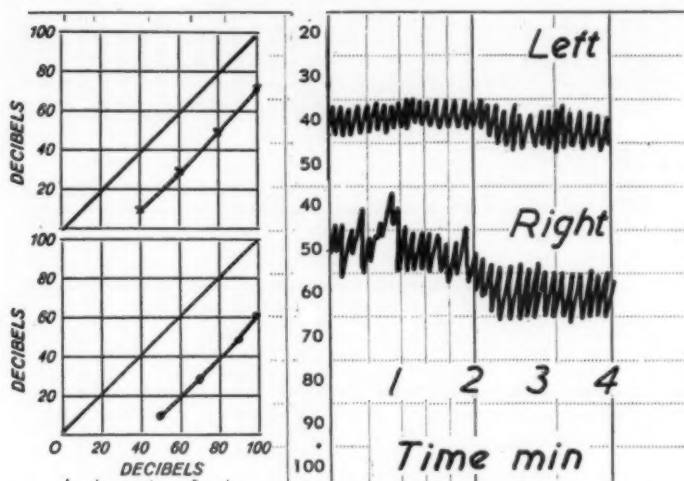


Fig. 3. Case E. L., farmer, age 58 years. The drums are normal. The audiograms demonstrated bilateral, moderate perceptive hearing loss in the 2000-6000 cps. range. The diagnosis was presbycusis.

The monaural loudness balance test between 3000 and 6000 cps. reveals the absence of loudness recruitment in both ears. The threshold tracings at 6000 cps. show a normal course in the left and a 10 db per-stimulatory loss in the right ear during sustained stimulation. The width of the excursions increases about 1 to 3 db in size during stimulation.

The deviations from the initial threshold value during continuous stimulation are not so clear cut. At two frequencies there was no significant change, but in one ear the threshold improved by 6 db and in three ears it dropped 6 to 10 db below the reference level. Because of the small number of cases it is not possible to make generalizations, but it seems obvious that some nonrecruiting perceptively deaf ears may exhibit sig-

nificant temporary threshold losses during sustained stimulation.

Two of the patients in this group had had audiometric studies made earlier. This did not seem to affect the behavior of the group in any way.

Recruiting perceptive deafness was further subdivided into three different categories on the basis of the recruitment functions. The first group includes all ears where the rise in the loudness function was asymptotic, in the second group are the cases where the line connecting various equal loudness points was a straight one; in the third group the recruitment was delayed at low intensities. This is in line with the various recruitment curves presented earlier by Harris, Maines and Myers.¹⁶

The group of asymptotic recruitment includes 14 persons, for whom the results are presented at 23 frequencies. At six frequencies the per-stimulatory threshold loss exceeded 5 db. In one case the loss attained the really high value of 27 db after four minutes' stimulation. In all others the tracing was within ± 5 db limits from the reference level; the average response, therefore, seems to be similar to that in a normal ear. The audiometric results in the exceptional case (U. M.) are presented in Fig. 4.

The straight line recruitment group consists of the results obtained for eight persons at nine frequencies. The per-stimulatory threshold shift was within normal limits in all these cases.

Delayed recruitment was demonstrated in five cases and the results are shown at nine frequencies. It appears that in one case (at two frequencies) the per-stimulatory threshold loss amounted to 8 db.

The width of the excursions during the first minute's stimulation showed significant individual variations even in the group of asymptotic recruitment. At 15 frequencies it varied from 1 to 5 db and at eight frequencies it varied from 5 to 12 db. It is apparent that the latter values cannot be used as criteria indicating the presence of recruitment, because the

same width of excursions occur also in the normal, conductive, and nonrecruiting perceptive group. About two-thirds of the frequencies tested thus agree with the recruitment tests proper.

The nine frequencies tested in the straight line recruitment group include only one where the width of the tracing was as

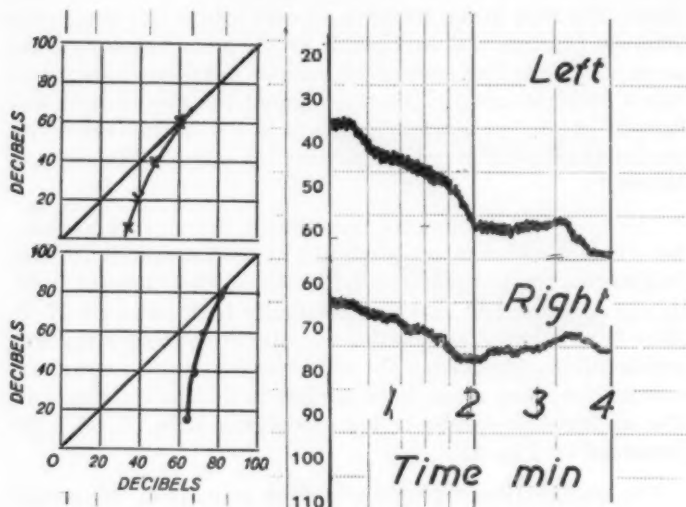


Fig. 4. Case U. M., business executive, age 36 years. The drums are normal. The audiograms revealed bilateral good hearing up to 3000 cps. and severe perceptive hearing loss in the higher range.

The monaural loudness balance between 3000 and 4000 cps. in both ears indicates asymptotic recruitment. The threshold tracings at 4000 cps. demonstrate a marked temporary threshold loss which is exceptionally large in the left ear. The width of the excursions is small and consistent with a recruiting deafness. It may be noted that the amplitude of the excursions decreases somewhat as a function of time.

small as 2 or 3 db. In all others the excursions were as wide as in any other nonrecruiting group.

Finally, at all nine frequencies where recruitment was initially delayed, all threshold tracings were larger than 5 db; therefore, on the basis of the threshold tests alone, one would not place them into the group of recruiting deafness.

The amplitude of the tracings remained stable on an average during four minutes' stimulation. In four cases there was a noticeable decrease (3 to 6 db) which placed the final width below the critical figure of 5 db, and these cases can thus be added to those in which conclusions regarding the presence of recruitment can be made from the threshold studies. Three of them were in the asymptotic recruitment group, one in the straight line recruitment group. It may be noted that in the ordinary Békésy audiogram the excursions were greater than

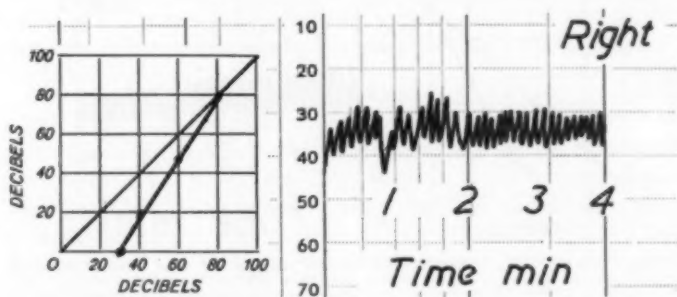


Fig. 5. Case J. S., medical student, age 23 years. The patient has tinnitus and a sense of fullness in the right ear; occasional slight giddiness. The drums are normal. The audiograms showed normal hearing in the left ear and 30-40 db perceptive low tone hearing loss in the right ear. The tentative diagnosis was Ménière's disease.

Binaural loudness balance test at 500 cps. shows a straight line recruitment. The four minutes threshold tracing at the same frequency pursues a steady course, and the width of the excursions remains unaltered. The amplitude of the tracings parallel those of normal ears in spite of complete recruitment.

5 db, corresponding to the initial width of the tracings with sustained one-frequency stimulation.

In only one case the tracings increased in size as a function of time. This case belonged to the group of straight line recruitment (S. L.) and the excursions were even primarily quite large, 10 to 12 db. In a per-stimulatory 80 db sensation level (above the threshold of the normal ear) balance test,¹¹ with a continuous tone in the impaired ear and an adjustable tone in the good ear, a five minutes' sustained stimulation presented a tracing pursuing a steady course.

In Figs. 5 and 6 are presented two cases as examples of the tests in the straight line and delayed recruitment groups.

About one-fifth of the recruiting patients had been previously tested audiometrically, and they all showed steady threshold excursions. Those with a clear decrease or increase in the width of the excursions were being tested by audiometry for the first time.

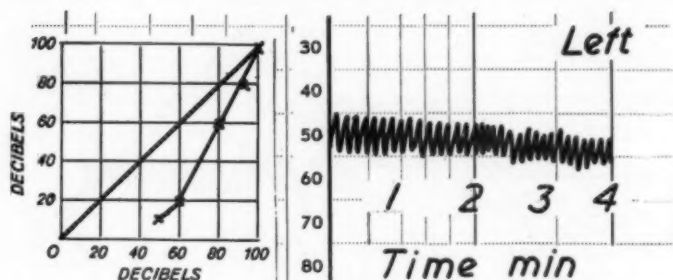


Fig. 6. Case V. V., industrial worker, age 54 years. The patient has had poor hearing in the left ear since infancy. The drums are normal. The audiograms revealed a nearly normal hearing in the right ear, and a marked, about 50-70 db perceptive, hearing loss in the left ear.

Binaural loudness balance test at 500 cps. shows delayed-straight line recruitment. The threshold tracing at the same frequency shows an equal, steady course and the width of the excursions remains unaltered. The threshold tests alone, without proper loudness balance, do not show that there is complete recruitment.

In addition, three patients, whose records are not included in this material, showed recruitment in the balance tests, though the threshold excursions remained between 20 and 30 db. An attempt was made during the following day to accustom them to the self recording technique, but this was unsuccessful. It is apparent that some slowly responding people will not give their true results in this type of test.

Occasionally one also finds some normally hearing and quite intelligent persons who do not succeed in the automatic threshold recording. In spite of repeated attempts the threshold variability remains between 15 and 30 db. On the other hand, there may be normally hearing subjects who present extremely small threshold excursions. One was included in the material used for another study;¹⁹ the excursions were at all frequen-

cies clearly below 5 db, and using interrupted tones, between 1 to 3 db through the whole range from 125 to 4000 cps.

DISCUSSION.

Because it is probable that per-stimulatory threshold shift in conductively deaf persons equals that in normals, only a few cases of this type of hearing loss were included. It seems that in both normal subjects and in those with conductive deafness there are frequently variations of around ± 5 db, which must be considered to represent the experimental error. The width of the excursions generally varies between 5 and 15 db, and only minor variations in this amplitude are likely to occur.

In nonrecruiting perceptive deafness there may be cases which show per-stimulatory threshold losses, in this study of the order of 10 db; however, a sufficient number of cases was not available for study, and later work may show some deviations from the above figure. For example, there were no cases of acoustic tumor, and in such ears in particular, Kos¹⁴ has demonstrated quite large threshold losses during sustained stimulation.

The width of the excursions may vary considerably, from 5 to 20 db. Small variations as a function of time can be demonstrated in either direction during sustained stimulation.

In the ears showing recruitment it is possible to obtain really large threshold losses during continuous stimulation. The results in the case presented in Fig. 4 are in agreement with those reported by Hood,¹² but it must be stressed that this was an exceptional case. In seven instances the threshold became worse by 6 to 10 db, and in the large majority of cases the threshold tracing followed the same steady course as in the normal or conductive group.

It must be stated, therefore, that although some large losses may occur the average response will be a steady tracing, or a tracing showing only minor evidence of fatigability. This is in accordance with previously published studies on above-threshold per-stimulatory adaptation tests,¹¹ and it demonstrates that, at threshold intensities, this phenomenon cannot

be used as a differential diagnostic auditory test. Even if Kos'¹⁴ report on auditory nerve fiber degeneration associated with large temporary threshold shift appears to be consistent with later control studies, the fact that extensive fatigability can appear in recruiting deafness, eliminates this test from differential diagnosis.

The division of the recruiting cases into various sub-groups was necessary to test the claim made earlier.^{2,15} viz., that the Békésy audiogram does not measure the IDL, but rather the variability around the threshold. Small variability indicates that the audibility increases rapidly with increasing intensity, larger variability is associated with normal or slower than normal function between the two.

We² stated that one would expect audibility to be intimately related to loudness. In the above groups, therefore, the smallest variability should occur in the asymptotic recruitment group. The straight line recruitment group may show normal, or less than normal variability, depending to some extent upon the threshold loss and upon the steepness of the recruitment curve. In the group of delayed recruitment the threshold tracings should parallel those of normal subjects.

It may be said that the same assumptions can also be made regarding the size of the IDL in these various groups, and that this neglected recruitment function is the cause of the greatly conflicting results of various IDL studies; however, before such an assumption is made, it must be shown that the number of discriminable steps is the same in a normal ear as in an ear with, for example, a 50 db threshold loss. The burden of getting this evidence is rather on the side advocating the use of IDL on the basis of that assumption.

It seems very hard to understand how a damaged end-organ could be capable of better intensity discrimination than a normal ear with all its delicate, unimpaired structures. It might well be that the discriminable steps in recruiting ears are as large, or larger than in normal ears and fewer in number. That would result in normal, or above normal IDLs; however,

if one should measure the IDL at the junction of this large step-wise change, one may get a very small intensity reading.

The results show that in most cases of asymptotic recruitment with rapid growth in audibility, the threshold variability is less than 5 db and indicates recruitment. Even in this group there are some cases in which the excursions are more than 5 db and parallel those in the other diagnostic categories. If the recruitment function is of the straight line type, more cases show normal threshold variability, and in the group of delayed recruitment practically all cases show normal excursions, between 5 and 15 db.

It seems clear that Békésy-type audiometry can never replace ordinary audiometry combined with Fowler's or Reger's balance tests. One is probably not misled in cases where small threshold variability is interpreted as to mean recruitment, but one misses equally many cases showing recruitment in the proper tests but failing to show it in the form of small threshold variability.

Of interest is also the variation in the width of the excursions as a function of frequency in recruiting deafness. This is apparent from the results in this paper, but it seems on the basis of a considerably larger material examined in this clinic, that the variability clearly decreases as the pitch increases. Thus, small threshold excursions are most readily demonstrated at 4000 or 6000 cps. Values indicating recruitment can also be quite easily obtained at 2000 or 1000 cps., but rarely if the frequency is further lowered; in fact, using the type of test procedure described in this paper, I have not yet found a single case showing 1 to 3 db threshold variability at 125 or 250 cps. If the threshold loss is considerable, one may get below 5 db at 500 cps., but in my experience even this is an exception rather than a rule.

This relation further weighs against the theoretical soundness of the IDL tests. If the width of the excursions depended solely on the diminished IDL, here would be an ideal area in which to demonstrate it. Even in the normal ear loudness increases for the low tones faster than at the middle frequencies,

and combined with low tone recruiting deafness, the rate should be quite fast and correspondingly the IDL very small. Experimental evidence, however, does not support this view.

The change in the width of the threshold excursions as a function of time is comparable to normal in the majority of recruiting cases. If there is a change during four minutes' stimulation, the variability is more likely to decrease than to increase. The reduction can be large enough to cause the case to be properly placed into the group of recruiting deafness although the ordinary Békésy audiogram, or the first one-minute single frequency tracing indicates greater than 5 db threshold variability. Hood's⁹ claim that the threshold excursions increase in size as a function of time in sustained stimulation, indicating the reversal of recruitment and a change-over from small to large IDL values, is not supported, and if there is any change it is generally a decrease.

Another major point should be stressed. It is apparent, even on the basis of the various recruitment functions, that it is quite a hopeless task to invent new tests with the object of revealing the presence or absence of recruitment at or close to threshold. This applies especially to the IDL tests, regardless of what one thinks of the soundness of the idea. It applies equally to Békésy audiometry, which as a rule seems to be reliable in cases where it indicates recruitment. If we want to invent new audiometric tests which reveal the presence of recruitment in all cases (gives the same information as Fowler's or Reger's balance test), then it is necessary to work at those intensities where even delayed recruitment becomes apparent. This means something like 60 to 80 db sensation level (re normal threshold), depending somewhat on the frequency and the threshold loss. Around this level practically all cases will show to which group they belong.

SUMMARY.

A short review is given of diagnostic recruitment tests at low sensation levels, *viz.*, IDL tests, per-stimulatory threshold fatigability and automatic threshold recording technique.

Data are reported on 39 patients belonging to various diagnostic categories. On the basis of the loudness balance functions the cases of recruiting perceptive deafness are further subdivided into asymptotic, straight line, and delayed recruitment groups. All cases were tested with alternate binaural judgment at the same frequency or monaural judgment between two frequencies. Automatic threshold recording was done with a Békésy-type audiometer providing intensity changes in 1 db steps at a rate of 140 db per minute.

The width of the threshold excursions was generally between 5 and 15 db in normal hearing persons. Exceptional cases can be found which exceed both these limits. The same applies to the conductive and nonrecruiting perceptive deafness. The majority of cases with asymptotic recruitment showed excursion amplitudes of less than 3 db. Most cases in the straight line recruitment group presented larger than 5 db excursions and practically all cases with delayed recruitment showed normal variability.

A per-stimulatory threshold shift of ± 5 db is considered normal. This also occurred in conductive deafness, while in the non-recruiting and recruiting perceptive deafness some ears showed a loss of the order of 10 db, and in some exceptions, more. It does not seem possible to use this test for subdividing the group of perceptive deafness.

The width of the excursions during sustained stimulation can undergo small variations as a function of time in each group. The majority show unaltered excursions, and if there is any change, it is likely to be a decrease from the initial values.

Finally, it is pointed out that any low sensation level test designed to substitute the loudness balance tests, is likely to fail in many cases. It is necessary to work on higher sensation levels if successful results are expected in all cases.

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THE PROBLEM OF POST-TONSILLECTOMY POLIOMYELITIS.*

An Analysis of 888 Cases of Poliomyelitis and 2,813 Tonsillectomies From a District in the Southwest
United States, 1949-1953.

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I. INTRODUCTION.

The possible causal relationship between tonsillectomy and poliomyelitis that develops shortly after operation has been recognized since 1910, but did not become a matter of general concern for another 20 years. In addition to this basic consideration, two other issues also began to be discussed as time passed—whether tonsillectomy predisposed not only to poliomyelitis but also to the more severe forms, and whether the removal of the tonsils at any period of life rendered the patient more susceptible to this disease.

The discussion has often, unfortunately, been attended by more heat than light. Experimental studies have not been conclusive. Statistical studies have been equally inconclusive. Sweeping conclusions have been drawn from isolated cases, or from series of cases too small to be of statistical significance. Indeed, a survey of the poliomyelitis-tonsillectomy literature permits one to believe, as he may wish:

1. That removal of the tonsils and adenoids in the so-called "polio season" multiplies the chance of contracting the disease, particularly in its more serious forms; or
2. That the development of poliomyelitis after tonsillectomy can be explained as merely fortuitous; or
3. That it can be explained as the result of exposure before operation.

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A survey of the literature also permits one to believe, as he chooses:

1. That removal of the tonsils increases the susceptibility to poliomyelitis, with the susceptibility extending over many years, if not for life; or

2. That the loss of these structures no more influences the chances of developing poliomyelitis than does the loss of the appendix.

Generally speaking, the literature of post-tonsillectomy poliomyelitis falls into three somewhat overlapping periods:

1. In the first period, running from 1910 to approximately 1930, only isolated cases or small groups of cases were reported. The first really extensive study was by Aycock and Luther,¹ in 1929. During this period the possible relationship between tonsillectomy and poliomyelitis was noted, but the discussion was usually tentative.

2. In the second period, which runs to approximately 1945 or 1946, much larger series of cases were reported, and the relationship was regarded as important.

3. In the third period, running from 1946 to the present time, a change in methods of reporting became evident. Observers began to report larger series of cases of poliomyelitis, some of which followed tonsillectomy. They also reversed the emphasis and showed the other side of the picture; that is, they began to record large series of tonsillectomies, some few of which were followed by poliomyelitis. During this period, experienced statisticians also began to analyze the records by sound statistical methods.

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There are a number of reasons why the argument over tonsillectomy as a predisposing cause of poliomyelitis has remained so confused. One of the most important, if also the least tangible, is the emotionalism apparently inherent in any discussion of this disease. Part of the responsibility for this situation must be attributed to the fund-raising activities of the National Foundation for Infantile Paralysis, valuable as the work of this organization has been. The publicity which attended the recent introduction of the Salk vaccine and the difficulties which promptly arose over its administration have done nothing to clarify the confusion. The single death during the testing period followed a tonsillectomy and occurred two days after the child had received the second injection of vaccine. The fact that poliomyelitis was already prevalent in the area, and the further fact that the child probably already had the disease when his tonsils were removed, have been lost sight of. The circumstances, as many otolaryngologists would testify, have brought some parents close to hysteria.

The chief reason, of course, why the possible tonsillectomy-poliomyelitis sequence has not been clarified is that we are still completely ignorant of many important facts about this disease. We know that the subclinical disease is far more frequent than clinical poliomyelitis, though the suggested ratios range from 1:100 to 1:600. We know that poliomyelitis is caused by a virus which is a total parasite and which has a specific affinity for neural tissue. We still know nothing really definitive about the mode of transmission. Many other immunologic factors are still obscure. We are not even certain that all the strains of the causative virus have been discovered. The developments of the last few years give hope that the answers to these and other questions will not be too long delayed. Until they are at hand, however, we can scarcely expect the disputed relationship between tonsillectomy and subsequent poliomyelitis to be completely clarified.

Meantime, the otolaryngologist will continue to be confronted with the necessity of performing tonsillectomy, particularly in young children, when the operation is indicated. No matter on what side of the argument he stands, he must bear in mind the possible subsequent development of poliomye-

litis and his possible responsibility for its occurrence, whether the disease was pre-existent, or fortuitous, or actually the result of the operation.

II. PURPOSE OF THE PRESENTATION.

An inclusive review of the literature of poliomyelitis would be an obviously impossible task. It has grown to monumental proportions and most of it either has nothing to do with tonsillectomy or mentions the operation only incidentally. The literature of post-tonsillectomy poliomyelitis is also large, but a selective review permits a record and evaluation of contributions that may throw some light upon this problem.

In addition to such a selective review, this presentation has another purpose; to put on record the tonsillectomy-poliomyelitis statistics for the period 1949-1953 in a community in the Southwestern part of the United States. The justification for the analysis is that only by the accumulation of data in adequate numbers from all parts of the country for periods of several years can this problem be resolved within the limits of our present knowledge.

It seems fair to say that the numerous general articles on post-tonsillectomy poliomyelitis represent no real contribution to the subject. For the most part, they amount to little more than expressions of the writers' opinions, secured from the data of other observers, or sometimes, from the unsubstantiated opinions of other writers. These articles contain no statistical evidence to support the points of view advanced, and for the most part, there is little evidence of the authors' clinical experience. They have been carefully reviewed in the preparation of this thesis, but for the most part they are neither recorded in the list of references nor commented upon further. The reviews of the subject which have appeared at intervals in the literature, such as Singleton's^{2,3} excellent analyses, are also omitted, since any material of value in them is utilized in the course of the presentation.

III. EXPERIMENTAL STUDIES.

It is now generally agreed that experimental poliomyelitis in the monkey differs sufficiently from the natural disease in

the human subject to make it necessary to be cautious in drawing firm conclusions from experimental studies;⁴ nonetheless, certain experimental observations which are pertinent to the subject of post-tonsillectomy poliomyelitis should be briefly mentioned:

Nasopharyngeal Transmission of Poliomyelitis.

In 1910, Flexner and Lewis⁵ inoculated the brains of monkeys with preparations of the nasopharyngeal mucosa of infected monkeys. No stool studies were made. The injected monkeys promptly developed the typical paralytic type of poliomyelitis, thus proving that the mucous membrane of the nasopharynx of the infected animals contained the virus of the disease. These animals were sacrificed, and a second set of monkeys was injected with preparations of their spinal fluids and brains. The virus was recovered in the nasopharyngeal mucosa of the injected animals. These observations permitted the conclusion that the virus of poliomyelitis may travel from the nasal mucosa to the brain and spinal fluid, or in the reverse direction.

In 1911, Landsteiner, Levaditi and Danulesco⁶ were able to produce poliomyelitis by submucosal injection of the virus into the tonsillar area. The following year Levaditi and Danulesco⁷ failed to produce the disease merely by rubbing or painting the tonsillar area with the virus, though application of the same preparation to the nasal mucosa produced typical disease. As Aycock⁸ observed, neither set of experiments proves very much, since "monkeys develop poliomyelitis with greater or less frequency resulting from numerous routes of inoculation."

In 1912, Flexner and Clark⁹ swabbed the nasal mucosa of a healthy monkey with a preparation of ground spinal cord secured from a recently paralyzed monkey. The treated animal was sacrificed at the end of 48 hours and suspensions of the olfactory lobes, the medulla, and various segments of the cord were injected into the brain and peritoneal cavity of three other monkeys. The animal injected with a suspension of the olfactory lobes died with paralytic poliomyelitis on the twelfth day. The other animals remained well. This experiment ap-

parently showed that the virus of poliomyelitis ascends by a direct lymphatic path from the nasal mucosa to the olfactory lobe before it reaches the medulla and cord, and that infection does not occur by way of the blood stream.

In 1938, Sabin⁹ reported a series of experiments on monkeys which may be briefly summarized as follows:

1. When preparations of the virus were applied to wounds created in the oropharynx by tonsillectomy, the monkeys remained well, whether the application was by swabbing, spraying, or nasal instillation.

2. When the virus was injected subcutaneously, intradermally or superficially into the post-tonsillectomy fossa, animals injected with preparations of low potency remained well. When larger quantities of a more potent preparation were injected, about a third developed the disease.

3. In this experiment, an attempt was made to re-create the conditions presumably present when the throat harbors the virus of poliomyelitis before tonsillectomy. Tonsillectomy was performed immediately after the tonsillar area had been injected with a preparation of the virus, in an endeavor to determine whether exposure of the cut nerve endings or cutting through tissue which contained the virus would stimulate the development of the disease. Only the animals in which the injection had been made deep to the bone developed poliomyelitis. The others remained well.

4. When the tonsillar area was injected before operation, as in the preceding experiment, and the fossa was also swabbed with virus immediately after the tonsillectomy, about a third of the animals succumbed to the disease.

These experiments showed several things:

1. In the monkey, the animal which most nearly resembles the human subject, the olfactory mucosa is the only known peripheral tissue which provides a portal of entry to the central nervous system by mere transitory contact with the virus of poliomyelitis.

2. Mere transitory contact between the virus and the normal

or injured pharynx or tonsils is not sufficient to produce the disease.

3. Poliomyelitis apparently develops after tonsillectomy when the virus is already present at operation, rather than as the result of contamination after operation. These observations support the concept that bulbar poliomyelitis which follows tonsillectomy within a period corresponding to the incubation period of the disease is preexistent. They do not support the concept that the disease is the result of postoperative infection.

Sabin's¹⁰ later observations on olfactory bulbs from patients with poliomyelitis, including studies of serial sections, failed to reveal the specific lesions which one might expect to find if the route of infection had been via the olfactory pathway. Previous investigators had considered the nasopharyngeal route the most probable natural portal of entry; more recent observers had considered only the olfactory mucosa of the nasopharynx as the route of infection. In Sabin's opinion, the tonsillopharyngeal route could not be regarded as the natural route. In fact, as he said, no direct data were available to show how the virus of poliomyelitis enters the central nervous system from the olfactory area or from anywhere else in the human body.

In 1939, using macacus cynomolgus monkeys, Burnet, Jackson and Robertson¹¹ produced the disease in seven of 19 animals by swabbing the tonsillopharyngeal region with poliomyelitis virus.

In 1942, Toomey and Krill,¹² after performing tonsillectomy on six macacus rhesus monkeys, kept the area between the cupped pillars flooded for five days with a 10 per cent suspension of poliomyelitis virus. As in Sabin's similar studies, the animals remained well, apparently because the contamination was entirely postoperative, was only superficial, and was accomplished with preparations of low potency.

In 1951, Faber, McNaught, Silverberg and Dong¹³ conducted a series of experiments on cynomolgus monkeys. All the animals were prepared by division of the olfactory tracts, to pre-

clude entry of the infection through the olfactory nerves. The results were as follows:

1. The tonsils were enucleated immediately after application of the virus to the pharynx. Paralysis ensued in four animals within seven to 10 days; in one instance it was limited to the bulbar and in three to the cervical innervations.

2. Tonsillectomy was performed three days after the virus had been injected into the thalamus. In this group of animals paralysis ensued five days after inoculation and two days or slightly less after tonsillectomy. The entire cord as well as the bulbar center was involved. The rapidity with which poliomyelitis developed in this experiment suggested to Faber and his associates that the physical stress of operation greatly enhanced the speed and severity, and to some extent the localization, of the infectious process. They believed that the results could be attributed to the introduction of the infection into the divided nerves before the endings could be covered or sealed off by blood. In an earlier publication, this group had not regarded the presence of "bare" nerve endings as essential to penetration of the virus.¹⁴

3. In this experiment the virus was applied to the pharynx, as in the first experiment; but, before tonsillectomy was performed, the area was swabbed with a 2 per cent solution of tincture of iodine. The iodine application was made 30 seconds after the application of the virus and 60 seconds before tonsillectomy. All six animals remained well. This method does not seem to have been tested in human subjects, nor is there any record of laboratory studies to determine whether tincture of iodine in this concentration will destroy the virus of poliomyelitis.

4. Control animals were treated simply by swabbing the surfaces of the mouth and pharynx with the virus; tonsillectomy was not performed. Five of the seven developed poliomyelitis, in one instance of the bulbar type.

In 1946, Faber and Silverberg¹⁵ had made a study of eight autopsied cases of poliomyelitis to determine the possible portals of entry of the virus. Their theory was that the primary invasion occurs through nerve fibers with peripheral endings

on mucous surfaces, the virus being conveyed in a central direction to the regional ganglia and thence to the central nervous system. Although they believed that entry could be effected via the mucosa of the nose, pharynx, trachea, esophagus, intestine and bronchus, they regarded the pharynx as a particularly favorable site, though their reasons for this point of view are not particularly convincing.

Neutralizing Properties of the Nasopharyngeal Mucosa.

In 1917, Amoss and Taylor,¹⁶ in a series of 56 experiments, showed that washings of the nasal and pharyngeal mucosa apparently possess definite power to inactivate or neutralize the active virus of poliomyelitis. The ability varied from person to person, and inflammatory conditions of the upper air passages tended to destroy or diminish it. In the opinion of these observers, their results explained why the olfactory bulb and nerve, along with their centers, are so seldom involved at autopsy in poliomyelitis.

In 1935, Lennette and Hudson,¹⁷ after sectioning the olfactory tract of five monkeys, sprayed the nasal cavity with a preparation of virus. All the animals remained well. When the same experiment was carried out on nine intact monkeys, all died of poliomyelitis. The five sectioned monkeys also remained well after intravenous injections of 10 cc. of 10 per cent preparations of the virus. When the same experiment was carried out on five intact monkeys, four died. Lennette and Hudson concluded from these studies that virus is excreted from the blood stream onto the nasal mucosa, whence it enters the endings of the olfactory nerve and then migrates to the central nervous system.

In 1939, experimental studies by Burnet, Lush and Jackson¹⁸ demonstrated only slight "virucidal" activity against the virus of poliomyelitis in the nasal secretions. The finding, however, was thought to be significant, and to offer a possible explanation for the infrequent changes found at autopsy in the olfactory nerve and its centers.

In 1941, Allen and Clark,¹⁹ working along the same line, studied the ability of soluble components of the olfactory mu-

cosa to neutralize the virus of poliomyelitis. Consistently positive results were not obtained.

IV. STATISTICAL STUDIES.

In 1942, after a review of the literature and a personal survey of his own, which will be commented on later, Seydell²⁰ remarked that it was impossible to draw any definite conclusions relative to the relationship of tonsillectomy and poliomyelitis from his own survey or from the literature. Much the same situation, as has already been intimated, still prevails.

On the surface, it might seem that nothing would be simpler than to construct a table which would show, for each report in the literature, such essential data as 1. the number of cases of poliomyelitis in the series; 2. the number of tonsillectomies performed over the same period; 3. the period covered by the study; 4. the number of cases of poliomyelitis which followed tonsillectomy; 5. the number of cases of the bulbar variety of disease in the total series; and 6. the number of post-tonsillectomy bulbar cases. The construction of a table that is complete in all these data is an impossible task. The table which attempts to present them (see Table I) has been permitted to stand, because it conserves space and avoids repetition. Actually, aside from the blanks which indicate data not stated, or so stated that they are not useful for tabular purposes, the presentation is misleading in a number of respects:

1. Only a very few of the reports contained the essential data just outlined. Reports that contained them all were exceptional.

2. In many instances complete data were lacking as to the tonsil status of a considerable number of patients, and sometimes of most patients. This information would seem essential to any discussion of the subject.

3. In only six of the 19 recorded series (see Table II) was there any attempt to supply the number of tonsillectomies performed over the period in which the cases of poliomyelitis occurred.

4. In many instances the studies were limited to special age

TABLE I.
 Poliomyelitis After Tonsillectomy. Essential Data From Selected Reported Cases.

Date, Authors	Period Surveyed	Location	Poliomyelitis Cases Bulbar	No.	Tonsillectomy		Remarks
					Bulbar	Yes Bulbar	
1929, Aycock and Luther ¹	1927-28	Vermont, Mass.	714	497	217
1937, Fisher and Stillerman ²²	1935	New York City	686	55	10	5 Within 1 month
1938, Eley and Flake ²³	Boston	418	131	40	194	91 17 in 1 month
1938, Fischer, et al. ²⁴	1937	Toronto	507	63	20	231	43 8 tonsillectomies 1937, 4 bulbar. Age 3-12.
1941, Top, Vaughan ²⁵	1939	Detroit	521	4.2	215	27%
1941, Helms ²⁶	1937-38	New South Wales	711	91	7	3 Within 7-22 days
1942, Seydell ²⁰	1940	Kansas	537	53	99	46 Within 1 month
1944, Lucchesi and LaBocetta ²⁷	1937-42	Philadelphia	432	76	25	164	51 Including bulbo-spinal type
1944, Howard ²⁸	1937-43	Cincinnati	257	6
1945, Anderson, J. A. ²⁹	1943	Utah	400	17	17 Within 1 month. Only summer months surveyed.

TABLE I—Continued.

Date, Authors	Period Surveyed	Location	Pollomyelitis Cases Bulbar	No.	Tonsillectomy Bulbar Yes	Remarks
1946, Roberts ³⁰	1946	Connecticut	180	49	56	5 Within 1 month. Only summer months surveyed.
1947, Pedersen ³¹	1941-45	San Francisco	492	206	11	148 24 11 in 2 months
1947, Winborn and Stansbury ³²	1946	Dallas	134	44%	5	66% 17 January-August surveyed.
1949, Cunnings ³³	1946	13 States	2,290	382	56	18 1-2 months.
	1947	23 States	4,331	509	24	6
	1948	36 States	10,624	1,481	38	27
1950, Anderson, G. W., et al. ³⁴	1946	Minnesota	2,709		37	16 1-3 months.
1952, Miller ³⁵	1951	Los Angeles	675	469	61	53
1952, Miller ³⁶	1949	Los Angeles	1,229		5	2 Within 1 month.
	1950	Los Angeles	984		8	
	1951	Los Angeles	1,388		7	
1952, Tops ³⁷	1940-49	Detroit	1,947	936	1,011	

* A number of reports were necessarily omitted from this table because the data were so stated that they were not useful for these purposes. No attempt has been made to derive data in any category by combinations of statistics or otherwise. Since the data stated were not always entirely clear, there is no doubt this table contains some errors.

groups, or to certain months of the year, the ages and months varying from report to report. The individual author has, of course, the right to set up the framework of his own study, but individual vagaries make cumulative studies practically impossible.

5. Some series include all patients who developed poliomyelitis after removal of their tonsils, regardless of the time interval. Other series give the information only for tonsillectomies performed one or two months before the poliomyelitis developed.

6. As has been suggested, the diagnosis is probably questionable in at least some of the cases of poliomyelitis, as well as the post-tonsillectomy cases. Only the report by Siegel and his group²¹ specifies methods of confirmation of the diagnosis in the post-tonsillectomy cases.

7. In a sense, much of the material studied is weighted for the reason that it is derived entirely from hospital cases. As a number of observers have pointed out, hospital facilities are overcrowded during epidemic years, and only patients with the most severe types of disease are admitted. This fact undoubtedly explains the abnormally high incidence of bulbar poliomyelitis in certain series.

8. The data in the tonsillectomy table (see Table II) are more accurate and certainly constitute a fairer presentation of the tonsillectomy-poliomyelitis sequence than the data in Table I. Table II, however, also suffers from certain defects. The first is that some of the material was collected by questionnaire. The returns were surprisingly good, but since the diagnosis, in effect, rests upon the patient's accuracy, this is still not the best way to secure an overall picture. The second defect is that tonsillectomies done in private offices are not included in recorded series. At first glance, this might seem a trivial omission. Frequently it is not. It was surprising to find that 23 of the post-tonsillectomy cases collected by Siegel and his group^{21,41} in New York City followed operations done in private offices. These cases are necessarily omitted in detailed analyses. Finally, because the data produced were obviously incomplete, some of the tonsillectomy series are esti-

TABLE II.
 Poliomyelitis After Tonsillectomy. Collected Series of Tonsillectomies.

Date	Author	Period Surveyed	Location	Tonsillectomy	Cases	Poliomyelitis Bulbar
1944	Page ²⁸	1937-39-41	New York City	8,915	1	0
1945	Anderson, J. A. ²⁹	1943	Utah	4,199	17	17
1946	Roberts ³⁰	1943	Connecticut	6,610	5
1946	Cunning ³⁰	1943-45	New York City	2,289	3	0
1947	Pedersen ³¹	1941-45	San Francisco	57,796
1948	Kinney ⁴⁰	1946-47	Ohio	9,873 12,413	237 184 1
1950	Anderson, G. W., et al. ³⁴	1946	Minnesota	7,820	37
1951	Siegel, et al. ²¹	1944-49	New York City	53,007	30	17
1952	Miller ³⁶	1949	Los Angeles	14,812	10
		1950	Los Angeles	14,886	8
		1951	Los Angeles	14,544	10

mates, made by competent statisticians, for the most part, but nonetheless estimates.

9. None of these data makes allowances, and for practical reasons cannot, for variations in the virulence of the poliomyelitis virus from year to year and from locality to locality. They also make no allowances for host resistance.

What all of this adds up to, to put it quite bluntly, is that as matters now stand, we have very little reliable information concerning the possible relationship of tonsillectomy and poliomyelitis. The comment, in fact, seems perfectly fair that many reports amount to little more than "speculative playing with numbers."

V. COMMENTS ON RECORDED SERIES.

Sheppard,⁴² in 1910, was the first to call attention to the occurrence of poliomyelitis after tonsillectomy. Thirty-one years later, in 1941, the simultaneous occurrence of five cases in siblings submitted to tonsillectomy on the same day^{43,44} focussed the attention of both profession and public on the possible relationship of operation and disease.

Sheppard's report was an epidemiologic study of 200 cases of poliomyelitis in Springfield, Mass. In two cases tonsillectomy had preceded the illness. In one case the operation had apparently been performed at the onset of the infection; in the other, bulbar poliomyelitis followed tonsillectomy within the accepted incubation period.

Aycock⁴ considers it interesting that Sheppard "intuitively" singled out tonsillectomy as a possible etiologic factor in poliomyelitis. His (Sheppard's) reasoning was that tonsil and adenoid tissue might offer resistance to microbial invasion. If so, he said, "it might seem on *a priori* grounds injudicious to remove an inflamed tonsil, especially in the presence of an acute epidemic infection characterized in many instances by involvement of the upper respiratory passage." Be this as it may, two instances of poliomyelitis in 700 tonsillectomies does not seem highly significant.

In the so-called "K" episode, as already mentioned, five chil-

dren living in Akron contracted poliomyelitis after tonsillectomies performed on the same day, and three of the five died. The sixth and youngest child, who was not operated on, did not contract the disease, and the parents also remained well.

Although the details of the epidemiologic study by Francis and his associates⁴⁴ are too numerous to be repeated here; the important fact is that all of these children had contacts with young relatives who had positive stools, and that there was also contact with a relative who later apparently had a mild case of poliomyelitis. In the preliminary report of this episode, immediately after it occurred, Krill and Toomey⁴⁵ drew no conclusions as to the cause of the illnesses. Francis and his associates,⁴⁴ in their epidemiologic study the following year, stated that the operations had precipitated the attacks.

One of the most remarkable features of this episode is that Akron in 1941 had been singularly free from poliomyelitis. There had been only two cases in the city preceding the experience of the "K" family, and the "K" children had had no contact with these patients. Only six other cases occurred after the "K" episode. The four oldest children underwent teeth extractions in addition to adenotonsillectomy; in the reports of the experience no special attention is paid to the additional stress and strain which these procedures undoubtedly caused. It is particularly regrettable that background information on the number of tonsillectomies performed in Akron in the summer of 1941 was not supplied in any of the reports. Incidentally, as this thesis is being completed, newspapers and radios are carrying reports of a family in the Middle West in which eight of the 10 children are ill with poliomyelitis; antecedent tonsillectomy is not mentioned in any of the news items.

The essential data in Tables I and II are self-evident and need no special comment. It should be pointed out, however, that the percentage of tonsillectomized cases varies from series to series, depending upon several considerations:

1. The age of the patient included in the series. This is particularly true of the earlier reports, which were made when tonsillectomy was a far more frequent operation than it is now.

2. The period of the year included in the survey. In some

series, the data are limited to the epidemic months. This makes a difference in the later reports, since in a number of hospitals, by agreement or direction, elective surgery was almost entirely discontinued at these times.

3. The number of cases of poliomyelitis which had to be excluded from the analysis because the tonsil status was not known. In some instances this necessity reduced the original statistics by a third or more.

The reported incidence for tonsillectomy varies rather widely. Miller²⁶ found it to be 1:135 of the Los Angeles population in the two-to-nine-year-old group. Kinney⁴⁰ found it 1:113 in a county in Ohio. Lederer and Grossman⁴⁸ mention a ratio of 1:75 for the total population of the county. Roberts³⁰ uses a figure of 2,000,000 tonsillectomies annually in the United States. Top and Vaughan²⁵ remark that the incidence of the operation varies with the social and economic status of the population surveyed.

Miller's²⁶ cumulative survey of poliomyelitis after tonsillectomy for the years 1949, 1950 and 1951 in Los Angeles and Los Angeles County illustrates a dispassionate, scientific approach to the whole problem. For this three-year period he reported 3,601 cases of poliomyelitis in an area population of about 4,000,000 persons. Twenty followed tonsillectomy (see Table I).

In 1949 the ratio of poliomyelitis to the two-to-eight-year-old population was 1:1,352. In 1950 the ratio was 1:1,275, and in 1951 it was 1:999. In 1949, the estimated number of tonsil operations in this age group was 14,812; the expected cases of poliomyelitis after tonsillectomy 10.9 and the actual number 10. In 1950, the figures were, respectively, 14,586, 11.5 and eight. In 1951 the figures were, respectively, 14,544, 11.5 and 10. In short, the actual and expected incidences are practically the same. Curiously, there were fewer post-tonsillectomy cases in the months when poliomyelitis was most frequent than there were in the remainder of the year.

Miller²⁶ also studied the tonsillectomy status of 675 patients with poliomyelitis in Los Angeles County (see Table II). His figures for tonsillectomy, 30 per cent, are in general agreement

with those of other writers, who set the incidence of tonsillectomy in youth at about a third. The rate of poliomyelitis after tonsillectomy thus seems to follow the rate of tonsillectomy in the general population. Bulbar poliomyelitis, however, appeared more often and was likely to be more severe in recently tonsillectomized patients.

Tonsillectomy Studies.

The first serious attempt to determine the incidence of poliomyelitis after tonsillectomy, instead of merely stating the number of tonsillectomized patients in series of cases of poliomyelitis, was made by Page,³⁸ in 1944, from the Manhattan Eye, Ear and Throat Hospital (see Table II). In 8,915 replies (out of 27,849 patients circularized, which is a very good response), he found that there had been only one instance of "infantile paralysis" within a month after tonsillectomy. Page's investigation covered the years 1937, 1939 and 1941, in all of which poliomyelitis was prevalent.

In 1951 Siegel and his associates⁴¹ carried out a similar investigation in the same hospital for the period 1944-1949. In the 6,524 replies which they received to their questionnaire they found that 30 cases of poliomyelitis, 17 of the bulbar type, had occurred within a month after operation.

Roberts,³⁰ in 1946, arrived at the relative incidence of post-tonsillectomy poliomyelitis by a series of calculations. If, he said, government records could be accepted, that 2,000,000 tonsillectomies are done annually, then 70,000,000 would have been done between 1910, when the relationship was first observed, and 1945. A review of the literature up to 1943 revealed 274 cases of post-tonsillectomy poliomyelitis, a ratio of one to each 255,474 operations. Since no case of post-tonsillectomy poliomyelitis had been reported over 18 years of age, the tonsillectomy figure should be reduced to 52,500,000, which would be a ratio of 1:191,605. On the assumption that 53 per cent of all tonsillectomies are done in the July-October period, the figures are further reduced to 27,825,000 and the ratio to 1:101,551. Since the average incidence of poliomyelitis in the general population, according to figures supplied by the Bridgeport Board of Health, is 1:3,250, the incidence in the general

population is still 31 times more frequent than after recent tonsillectomy, even assuming that all 274 cases reported after tonsillectomy occurred in the so-called poliomyelitis months (which they did not). It is doubtful that a statistician would be willing to accept this reasoning, but the calculations are cited for what they are worth, as an illustration of one kind of attempt that has been made to reach a conclusion in this matter.

VI. CONCLUSIONS FROM STATISTICAL SURVEY.

Conclusions drawn from these statistical surveys are, naturally, widely divergent. Some of them seem justified by the studies on which they are based; others do not. Certain of them may be mentioned:

The earliest series of cases reported from the special viewpoint of a poliomyelitis-tonsillectomy relationship was by Aycock and Luther,¹ in 1928 (see Table I). From this investigation, they concluded that it could not be stated with certainty that the preceding tonsillectomy was responsible for the localization of the paralysis. In 1942, however, after a survey of the literature and a study of additional data, Aycock⁴ concluded that a causal relationship does exist between the removal of the tonsils and the occurrence of poliomyelitis within a specific interval. He also noted that this concept was in line with experimental observations, which showed that bulbar poliomyelitis was likely to occur after tonsillectomy when the virus was already present at the time of operation. His data also showed that bulbar poliomyelitis was more frequent at all ages when the tonsils had been removed. He was not willing to conclude that the absence of the tonsils predisposed to clinical poliomyelitis, even though tonsillectomy had been performed more frequently in patients with poliomyelitis than in controls studied in the same area. Aycock felt that because of numerical considerations (the fact that roughly 30 per cent of the population undergoes tonsillectomy at some time, whereas poliomyelitis occurs in only a fraction of 1 per cent), it would be difficult to say to what extent the operation should be avoided because of the relatively small risk of developing bulbar poliomyelitis. The risk should be recognized, but the

decision for or against operation should be upon the basis of the indications in the individual case.

Toomey and Krill,⁴⁶ who reported a combined series of 430 cases, from Akron and Cleveland, concluded that more patients contracted bulbar poliomyelitis after tonsillectomy than could be explained by mere chance or random sampling. From the standpoint of the relationship of the mortality to the tonsil status, it seemed to make little difference whether the tonsils were in or out; in fact, if mortality were the only criterion, it would seem that death was less apt to occur in previously tonsillectomized patients.

Siegel and his associates,^{21,47} by statistical methods, determined that the risk of contracting poliomyelitis was significantly greater than the expected risk within a month after operation, but observed no increase in the incidence of bulbar paralysis after six months had passed. On the other hand, differences in the numbers of observed and expected cases were often not great enough to rule out sampling variations, while in other instances the figures were at the borderline of statistical significance. This group considered children up to nine years of age more vulnerable to poliomyelitis when they were tonsillectomized in the spring, especially in June, and regarded January, February and March as the safest period of elective surgery in New York.

Langworthy,⁴⁷ while he would naturally be unwilling to operate in the midst of an epidemic of poliomyelitis, who provided no independent statistical data, took rather violent exception to the assumption of a tonsillectomy-poliomyelitis relationship. He saw no justification for postponing tonsillectomy when it was needed because a few cases of poliomyelitis existed in the community. He mentioned the harm that delay might do to children with bad tonsils, malnutrition, repeated aural and upper respiratory infections, and infections of the cervical glands.

Wesselhoeft,⁴⁸ who also provided no supporting data, had originally refused to perform tonsillectomy during the Summer months. By 1948 he had changed his mind and felt that in the New England states the ideal time for the operation was

immediately after the close of school. He would continue to operate through the Summer if no cases were reported from the community and there was no known possibility of exposure from the outside. He justified his stand, in part, by other dangers, such as upper respiratory infections, when the operation was performed during the Winter.

Miller,³⁶ whose sound statistical surveys have been quoted at considerable length, concluded that during the years of his study the actual number of cases and the expected number were so close to each other that the differences were of no significance and tonsillectomy could be assumed to have played no part in the development of the disease during the years in question. In his survey of the 1949 material Miller mentioned that twice as many children had undergone tooth extraction as tonsillectomy. He felt that there was nothing in the figures to indicate that tonsillectomy need be discontinued unless one happened to believe that its performance any number of months before the onset of the infection could be a factor in its development.

Cunning,^{33,39} whose yearly surveys in 1947, 1948 and 1949 were conducted with great care, and were as nearly nationwide as he could make them, did not feel that his collected data justified a statement of a causal relationship between poliomyelitis and tonsillectomy. He realized that he was in disagreement with many of his associates when he took this position. In his own opinion, acceptance of a predisposition to the disease after tonsillectomy was based in part on experimental evidence and in part on limited clinical evidence and surveys. He did not think that tonsillectomy should be postponed indefinitely merely because poliomyelitis is prevalent in the Summer months, though as a matter of common sense, elective surgery should be discontinued promptly if there is a rise in the incidence. Unless conclusive evidence is forthcoming as to the relationship, the otolaryngologist should decide whether hardship to the patient by delay and postponement is greater than the still undetermined risk of increasing his susceptibility to poliomyelitis by performing a tonsillectomy. This is a reasonable and sound position, based on a wide clinical experience

and supported by ample statistical data, personally collected and surveyed.

VII. CONCEPTS OF THE TONSILLECTOMY-POLIOMYELITIS RELATIONSHIP.

In 1917, on the basis of his own experience and research, Seydell⁴⁹ took the position that the tonsils were a harbor of infection in poliomyelitis. Like Rosenow, he believed that tonsillectomy in the course of the disease was helpful. In this same communication Seydell cited the studies of Vaughan, Vaughan and Palmer:⁵⁰ During the 1916 epidemic in New York, a large number of the children affected had hypertrophied tonsils, or adenoids, or both. The disease was uncommon when the tonsils had been removed. After the epidemic, in which 8,928 cases occurred, an investigation in the public schools showed that not a single case had occurred in 1,404 tonsillectomized children, though in 18 instances there has been cases in the same family, and in 93 cases in the same house. The concept of a possible protective effect of tonsillectomy has, of course, long since been discarded.

Since the virus of poliomyelitis travels along the nerves, and since it may have an obligate affinity for the gray fibers of either medullated or unmedullated nerves, it may be that when the axis is exposed during tonsillectomy, the virus is more easily absorbed.⁴⁶ In the healing process, new vascular networks are laid down, accompanied by the deposition of new unmedullated fibers, which readily absorb the virus. On the other hand, most pathologists believe that by the end of 24 hours after tonsillectomy, a local tissue reaction has developed which would prevent the invasion of most pathogenic organisms.

To Seydell⁵⁰ it seems more unlikely that the virus can penetrate the scar tissue which develops in the tonsillar fossa after tonsillectomy than that it can invade the loose lymphatic tissue of the tonsil and its lymph and blood vessels.

Aycock⁴ discussed the possible relationship of poliomyelitis and tonsillectomy from the standpoint of what he calls autarcesis, that is, resistance to the host, independent of immunity

from exposure to the virus. He believes that the selectivity of the development of bulbar poliomyelitis after tonsillectomy is best explained in this manner, on the assumption that some added circumstance plays a part in determining whether clinical or subclinical disease will follow exposure to the virus. This selectivity, and the relatively more frequent occurrence of bulbar disease in tonsillectomized persons, in his opinion, comprise a demonstration that the nasopharyngeal mucosa may be the *locus* of at least one added circumstance that determines the outcome of exposure. His conclusion is that since tonsillectomy is practically always elective as to time, changing the season when it is done so that it will not coincide with the season of poliomyelitis prevalence, could eliminate many cases of a highly fatal form of the disease.

McCormick⁵¹ postulated a possible relationship between the basic hypertrophy of tonsils, adenoids and other lymphatic structures often present before tonsillectomy and the rather general lymphatic hypertrophy frequently present in poliomyelitis. He linked up this observation with the lymphatic hypertrophy frequently found in nutritional deficiency, especially vitamin-B deficiency. In the 1937 epidemic in Toronto it was found that among persons on relief, who had a low vitamin-B intake, there were four times as many cases of poliomyelitis as in the rest of the population.

McCormick also mentions other possible factors, aside from a direct tonsillectomy-poliomyelitis relationship, including the shock of the operative procedure, and the anoxia produced by anesthesia and the barbiturates often used in preoperative preparation. He marshals considerable evidence for this theory, including the fact that brain tissue deficient in vitamin-B absorbs oxygen more slowly than normal brain tissue, and the further fact, demonstrated by Van Liere, that severe cerebral anoxia can produce an ascending type of paralysis, with eventually, bulbar and cerebral involvement. McCormick's conclusion is that neuromuscular anoxia, associated with either a deficient nutritional status, or surgical anesthesia, or both, may supply the missing link in the etiologic relationship between tonsillectomy and poliomyelitis. In his opinion, the association may be much closer than ordinary contact.

Several observers, notably Top and Vaughan,^{25,27} believe that factors other than the tonsillectomy, with its surgical wound, may play some part in the development of poliomyelitis after operation. They mention stress and strain, trauma, physical activity, the body type, endocrine changes, dietary deficiencies, pregnancy, and preceding disease. Cunning³⁰ argues along much the same line. He assumes that in the large number of persons who harbor the virus of poliomyelitis with few, if any, clinical manifestations, there is some unknown factor or phenomenon which maintains an equilibrium between the host and the virus, so that clinical disease does not develop. He further assumes that any disturbance which upsets this equilibrium may so accelerate the action of the virus as to provoke clinical disease. If tonsillectomy is regarded as such an activating factor, then one must also take into account the attendant surgical shock and the effects of anesthesia, both of which are present in all surgery, regardless of the nature or the site. Since tonsillectomy is the procedure most often performed upon children of the age group most susceptible to poliomyelitis, the emphasis has been put upon it rather than upon the surgical shock and anesthesia associated with all operations. In other words, if tonsillectomy has any influence in the development of poliomyelitis, it is probably by lowering the resistance of the body rather than by the existence of a surgical wound in a particular location.

VIII. SURVEY OF THE TONSILLECTOMY-POLIOMYELITIS SEQUENCE IN A SOUTHWESTERN COMMUNITY.

Materials and Methods.

This survey was conducted in a community in the Southwest part of the United States in which poliomyelitis has been epidemic in several recent years, but in which no previous survey has been conducted to determine the possible relationship between the disease and previous tonsillectomy.

This community, which has a population of approximately 65,000, is located in a district of 260,000 persons. Medical practice comes from a large adjacent portion of this total area, and physicians in the whole district refer many of their difficult cases to the community.

The data were derived from two sources: the record libraries of the two local hospitals which treat poliomyelitis and, when the information contained in the records was not complete, from personal letters to the patients or their parents.

These letters explained that the objective of the study was a determination of the possible relationship between tonsillectomy and poliomyelitis. They requested that the writer be informed, on an enclosed card, whether the tonsils had been removed before the development of the disease, a simply worded inquiry which needed only a *yes* or *no* answer. The investigation thus avoided the pitfall inherent in inquiries which concern the development of poliomyelitis after operation. In cases which had ended fatally a carefully worded paragraph replaced the inquiry used otherwise.

The investigation covered the five-year period, 1949-1953, inclusive, and thus included both epidemic and non-epidemic years.

Essential Data.

The essential data in this investigation (see Table III) are as follows:

TABLE III.

Distribution of 888 Cases of Poliomyelitis According to Tonsil Status in Community in Southwest United States 1949-1953.*

	Total Cases	Tonsils In Situ	Tonsils Removed	Status Unknown
Total Cases	888	534	254	100
Spinal	767	468	211	88
Bulbar	121	66	43	12
Deaths	11	4	4	3
Spinal	2	1		1
Bulbar	9	3	4	2

* During this same period, 2,813 tonsillectomies were performed in this community.

In all, 888 cases of poliomyelitis were treated in the two hospitals in this community during the period 1949-1953. In 100 cases information as to the tonsil status could not be secured, and the analysis, therefore, chiefly concerns 788 cases, the essential facts of which are as follows:

In these 788 cases, 534 patients, 67.7 per cent, had their tonsils *in situ* when they contracted the disease.

Two hundred fifty-four, 32.3 per cent, had had their tonsils removed at some time in the past.

No patients in the series had undergone tonsillectomy within a month of their illnesses.

In 121 of the entire series of 888 cases the poliomyelitis was of the bulbar type. In 12 of the 121 cases no information could be secured as to the tonsil status of the patients.

In the remaining 109 cases of bulbar poliomyelitis, the tonsils had not been removed in 66, 60 per cent, and had been removed in 43, 40 per cent.

There were 11 deaths in the total series, four in patients whose tonsils had not been removed, four in patients who had undergone tonsillectomy, and three in patients whose status was unknown. This is an unusually low case fatality rate.

No patient in the series contracted poliomyelitis within a month of tonsillectomy. One patient, five years of age, became ill six weeks after the removal of his tonsils (1949), and another, nine years of age, six months after tonsillectomy (1950). Both patients had the spinal type of disease, and both recovered completely.

There were no instances of bulbar poliomyelitis within a six-month period after tonsillectomy.

During this same five-year period, 2,813 tonsillectomies were performed in the two hospitals surveyed. In only two instances, as just noted, did poliomyelitis develop within a six-month period after the operation, and in no instance, also as just noted, did the bulbar type of poliomyelitis occur within this postoperative period.

Yearly and Seasonal Distribution of Poliomyelitis.

The five-year seasonal distribution of the 888 cases of poliomyelitis surveyed in this study (see Fig. 1) is typical of the seasonal prevalence of the disease in this part of the United States. It is most frequent from May through September, but

a scattering of cases occurs through the remainder of the year. This figure (see Fig. 1) also shows the yearly and monthly distribution of the 2,813 tonsil operations performed over the same five-year period. It will be noted that more tonsil operations are done in this community in April, May and June than at any other period of the year. The explanation of the smaller

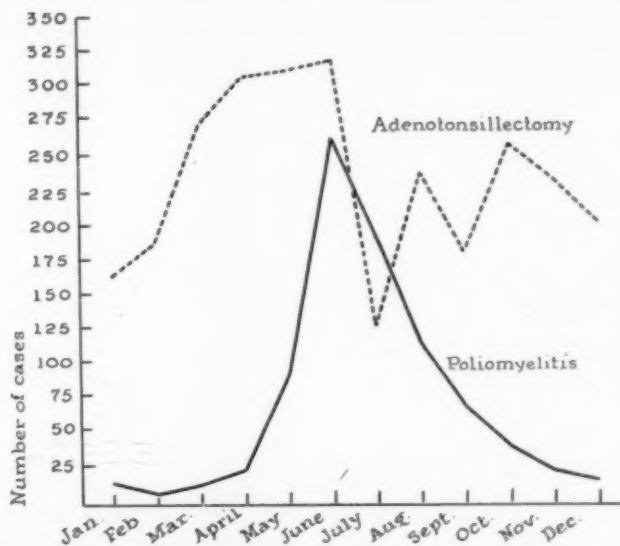


Fig. 1. Seasonal distribution of 788 cases of poliomyelitis and 2,813 adenotonsillectomies in a community in the southwest United States 1949-1953.

number of cases performed in July is the rather general practice, on the part of both profession and patients, of taking vacations during this month. The decrease is not due to discontinuance of tonsil operations in July. Even during the epidemic years of 1949 and 1950, particularly in 1950, throat surgery continued to be performed.

Age Incidence.

The age incidence in these 888 cases of poliomyelitis (see Fig. 2) shows nothing unusual, and is in correspondence with

observations from other parts of the country. Poliomyelitis is pre-eminently a disease of youth, and is most frequent in the five-to-10-year period, as this figure shows. Both cases of poliomyelitis after tonsillectomy occurred within this age group, but the number is too small to be worth showing graphically.

Hospital Incidence.

The distribution of all poliomyelitis cases in which the tonsil and adenoid status was known is shown separately for the two

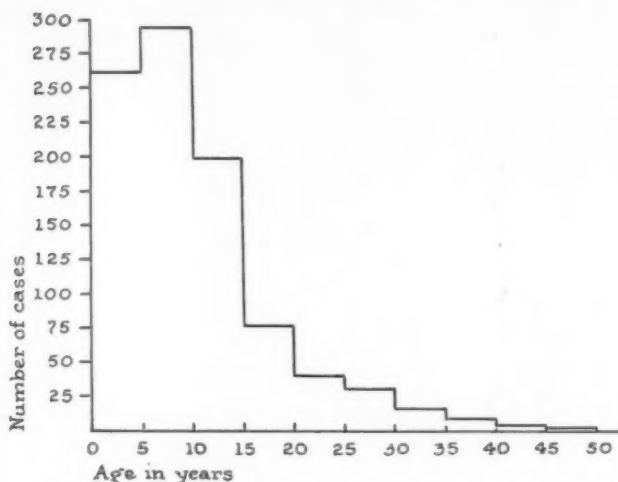


Fig. 2. Age incidence in 788 cases of poliomyelitis in a community in the southwest United States 1949-1953.

hospitals surveyed (see Table IV) chiefly as an illustration of how misleading conclusions based on small groups of figures can be. Only 92 of the 788 patients were treated in hospital "A". When this small group of cases is broken down by year and type of disease, the statistics are totally insignificant and some of the proportions are out of line. In this hospital, for instance, 38 per cent of the patients had had tonsillectomies sometime in the past. In hospital "B" the proportion was 31.4 per cent, and for the whole series it was 32.3 per cent. In hos-

TABLE IV.
Distribution by Hospital, Year, and Type of Disease in 788 Cases of
Polioomyelitis with Known Tonsil Status.

Tonsil Status	Hospital "A"		Hospital "B"		Both Hospitals	
	Spinal	Bulbar	Spinal	Bulbar	Spinal	Bulbar
1949						
In situ	25	2	233	35	258	37
Removed	5	6	90	9	95	15
						295
1950						
In situ	6	0	93	12	99	12
Removed	9	1	45	10	54	11
						111
						65
1951						
In situ	5	0	21	10	26	10
Removed	1	0	14	2	15	2
						17
1952						
In situ	13	2	58	2	71	4
Removed	7	3	33	7	40	10
						75
						50
1953						
In situ	3	1	11	2	14	3
Removed	0	3	7	2	7	5
						12
Total						
In situ	52	5	416	61	468	68
Removed	22	13	189	30	211	43
						534
Total	74	18	605	91	679	109
						788

pital "A," 13 of 35 patients without tonsils contracted bulbar poliomyelitis, 37 per cent, well over twice the estimated national average of 15 per cent, but readily explained by chance in so small a group of cases. In hospital "B", 30 of 219 patients without tonsils had bulbar poliomyelitis, 13.6 per cent, and the proportion for the total series was 17 per cent, well within the general average in hospital "B", and very close to it for the whole series. Entirely erroneous conclusions might be arrived at if only the figures for hospital "A" were used. The larger figures for hospital "B" and for the combined hospitals show again that there is no relationship between tonsillectomy and poliomyelitis *per se*, nor, in this series in this part of the United States, for the period 1949-1953, any clearcut relationship between tonsillectomy and the bulbar form of the disease.

Why the incidence of bulbar disease should be higher in non-epidemic years, such as 1951 and 1953, than in epidemic years is not clear. The most obvious explanations are that only small groups of cases are involved, and that the infecting virus in those years may have been of unusual virulence.

Comment.

There is really nothing more to be said about this series of cases. It would be possible to present the figures from other various aspects, but nothing would be gained by it. The essential fact is that over a five-year period, in 788 cases of poliomyelitis in which the tonsil status was known, and in 2,813 tonsillectomies performed over the same period, only two patients contracted the disease after tonsillectomy; in neither instance earlier than six weeks postoperative, and in neither instance of the bulbar variety. The fact that tonsillectomies were performed routinely over this period, without regard to whether or not poliomyelitis was epidemic in the community, is a fact of special significance in the argument over the possible influence of previous tonsillectomy on post-tonsillectomy disease.

It is true that the case fatality rate in tonsillectomized patients with bulbar poliomyelitis, four of 43 cases, was considerably higher than the rate in patients with tonsils *in situ*,

four of 66 cases. On the other hand, bulbar disease is always more serious than other varieties, and the number of cases in both tonsillectomized and non-tonsillectomized groups is too small to warrant conclusions as to risk.

IX. SUMMARY AND CONCLUSIONS.

1. A review of the literature dealing with the possible relationship of tonsillectomy and post-tonsillectomy poliomyelitis reveals two schools of thought: one group of observers believes that recently tonsillectomized patients are more susceptible to the disease, particularly the bulbar type of disease, because the virus of poliomyelitis has a readier route of entrance through the surgical wound, with its exposed nerve endings. The other school believes that tonsillectomy has no relationship to poliomyelitis which develops after it. In the opinion of this group, some cases of poliomyelitis which follow tonsillectomy are fortuitous, and others can be explained by such factors as stress, strain, surgical shock and the anoxia of general anesthesia, which are risks to be assumed in all surgery. The virulence of the causative virus is also regarded as an important consideration.

2. While the literature on the subject is extremely confused, it seems fair to say that those who believe in the causal relationship between tonsillectomy and poliomyelitis have based their opinions chiefly on experimental evidence, isolated cases, small series of cases, or series studied without due regard to statistical methods. Those who deny the relationship have investigated larger series, have studied them by sound statistical methods, and have also studied a side of the picture that was formerly ignored, namely, the incidence of poliomyelitis following large series of tonsillectomies.

3. It is unfortunate that the emotionalism inseparable from such a devastating disease as poliomyelitis has entered the picture and confused both public and profession. It has also placed a particularly heavy responsibility upon the otolaryngologist, who must decide, in the individual case, whether to perform an indicated adenotonsillectomy or to omit it for fear of the development of poliomyelitis.

4. The experimental literature is often confusing and frequently inconclusive, aside from the fact that natural and experimental poliomyelitis differ sharply from each other in some respects.

5. While a review of the clinical and statistical literature reveals widely differing viewpoints, the most valid studies indicate that the possible relationship between tonsillectomy and poliomyelitis remains to be proved.

6. An investigation of 888 cases of poliomyelitis and of 2,813 adenotonsillectomies performed over the same five-year period (1949-1953) in an area in the Southwest United States confirms the opinion that there is no causal relationship between tonsillectomy and post-tonsillectomy poliomyelitis. Only two cases of poliomyelitis occurred after tonsillectomy in the 788 patients whose tonsil status could be determined. The incidence of poliomyelitis after tonsillectomies performed during this period is thus entirely insignificant. Neither of the two cases occurred within a month of the operation, and neither was of the bulbar type. In fact, none of the 109 patients with bulbar poliomyelitis had undergone tonsillectomy within a six-month period.

7. This survey shows clearly the fallacies and pitfalls of basing conclusions upon single years and small series of cases from small institutions. When the figures in this series were broken down in this fashion, the conclusions reached were sometimes contrary to the conclusions arrived at for the total series and for the entire five-year period.

8. The soundest position in this matter was expressed by Cuning³⁸ in 1949, as follows:

a. A belief that tonsillectomy is responsible for the subsequent development of poliomyelitis is based on experimental evidence and limited clinical evidence and surveys.

b. Since there is no way of foretelling when an outbreak will occur in any community, tonsillectomy should not be postponed indefinitely merely because poliomyelitis is prevalent in the summer months. Naturally, if the incidence rises, all

elective surgery, including tonsillectomy, should be discontinued.

c. Until more conclusive evidence is forthcoming as to a causal relationship between tonsillectomy and post-tonsillectomy poliomyelitis, the otolaryngologist must decide, in each case that he encounters, whether the hardship to the patient by delay and postponement would be greater than the still undetermined risk of increasing his susceptibility to poliomyelitis by going ahead with tonsillectomy when it is indicated.

d. In short, until convincing proof is adduced that tonsillectomy increases the susceptibility to poliomyelitis, the solution of the problem is the application of common sense to the individual case: when poliomyelitis is prevalent in a community, elective surgery should not be performed. When it is not prevalent, tonsillectomy may safely be performed in the cases in which it is indicated.

9. The best way to settle this problem, in the light of present knowledge, would be to adopt Cuning's proposal that some national body undertake a controlled study, directed toward the future and not to the past, to determine what happens after tonsillectomy as well as how many tonsillectomized persons develop poliomyelitis. The responsibility of excessive oxygen administration for retrolental fibroplasia in premature children has just been settled by such a survey. It would be a far simpler matter to set up such an investigation of post-tonsillectomy poliomyelitis.

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TUMOR OF THE GANGLION NODOSUM OF THE VAGUS NERVE.*

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Although tumors of the carotid body have been recognized for many years, only recently have similar tumors arising in other sites in the neck been described. The following case is reported because multiple tumors were removed from the same patient, and also because of an apparent familial relationship, and finally because of the rarity of tumors of the ganglion nodosum.

REPORT OF CASE.

A white woman, single, age 25, was admitted to Detroit Memorial Hospital in June, 1951. She had noticed a small lump in the right side of her neck one year previously. The tumor had increased in size during the year but had caused no symptoms.

Past history was non-contributory. There was, however, an interesting family history. A sister of the patient had a carotid body tumor removed in November, 1947, at the age of 26 years. The tumor was reported to be a "malignant tumor arising in a carotid body." There was, however, no recurrence during the following three years, and review of the pathologic sections revealed the tumor to be probably benign. The patient had three brothers without tumors. Her mother and father were living and well. One uncle had died reportedly of carcinoma of the thyroid, and one of leukemia. The two uncles had six children, all without cervical tumors. An aunt died of diabetes at the age of 55 years.

Physical examination of the patient revealed a firm, smooth, non-tender nodule, 2 cm. in diameter in the right neck, which could be moved laterally, but not up and down. Heart and lungs were normal; blood pressure was 112/65 mm. Blood count and urinalysis were within normal limits.

On June 5, 1951, under local anesthesia, a tumor of the right carotid body was removed. The blood supply seemed to come from a point 0.5 cm. above the bifurcation of the common carotid, from the medial aspect of the external carotid artery. The tumor was dumb-bell shaped, measuring 22x19x17 mm., and was a typical carotid body tumor. There was no metastatic tumor in a lymph node removed from the fat, anterior

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to the carotid sheath. The postoperative course was uneventful, except for hoarseness, which slowly improved.

The patient was again admitted to Detroit Memorial Hospital on June 7, 1954, with a history of a lump in the left side of the neck for 18 months. She stated that when she was tired she "felt a strain" in the region of the lump and sometimes felt a thumping in it.

The patient had married 18 months previously, and had a child four months old. The lump in her neck had increased in size during the pregnancy.

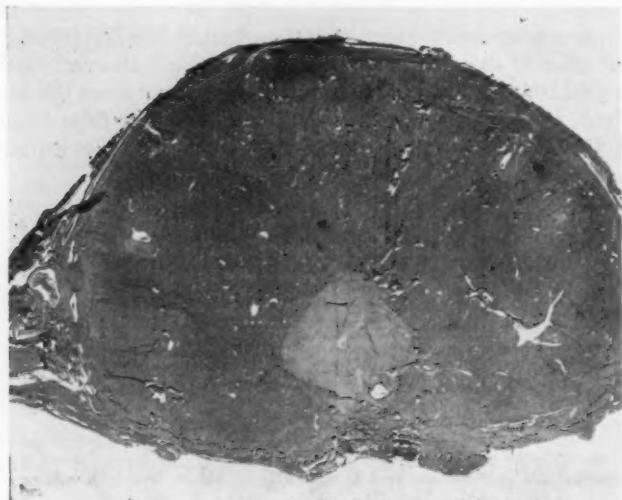


Fig. 1. Tumor of ganglion nodosum (x6).

On physical examination a mass was palpated in the left neck, anterior to the sterno-cleido-mastoid muscle, and at the angle of the jaw. As with the previous tumor, the mass could easily be moved laterally, but less freely in a vertical direction. Aside from the scar of the previously removed tumor, physical examination was negative. Urinalyses, serologic tests for syphilis and blood counts were normal.

On June 8, 1954, under endotracheal anesthesia, the tumor was removed. It was found to be a fusiform swelling of the left vagus nerve, directly posterior to the bifurcation of the carotid. The tumor was inseparable from the vagus, which was cut above and below the tumor. For a short period during the operation blood pressure readings could not be obtained.

The postoperative course was uneventful and the patient was discharged from the hospital on June 14, 1954.

PATHOLOGY REPORT.

The fusiform tumor measured 22x16x16 mm., with the vagus nerve projecting 4 mm. from each end of the tumor. The cut surface bulged slightly, and was pale pinkish-tan.

On histologic examination the tumor lay within the vagus nerve, with bundles of myelinated nerve stretched over the

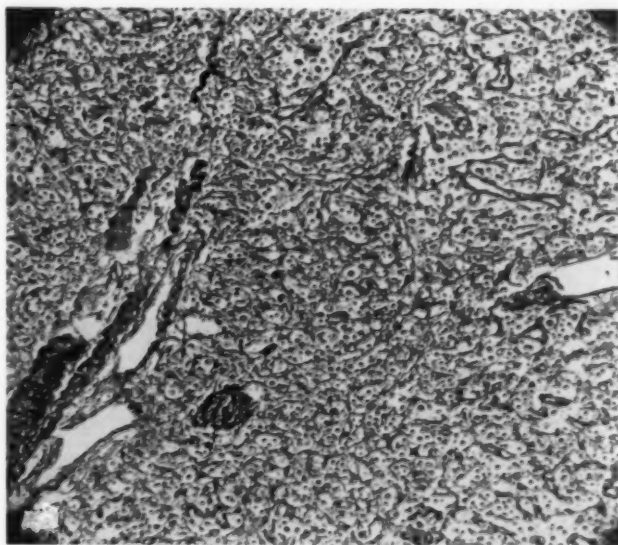


Fig. 2. Tumor of ganglion nodosum. Reticulin stain showing grouping of cells in acini and cords and intimate relationship to veins (x200).

tumor (see Fig. 1). The latter was composed of rounded groups and cords containing four to ten cells surrounded by reticulin fibers (see Fig. 2). Numerous small capillaries with occasional larger veins coursed in the scanty connective tissue between cell groups. In several areas larger collagenous bundles were found in the tumor. The nuclei of the tumor cells were finely granular and varied considerably in size; a few nuclei were over 30 microns in diameter (see Fig. 3).

Many cells had distinct nucleoli, but there were no mitotic figures.

The cytoplasm of the cells was often finely vacuolated. Occasional cells with pycnotic nuclei and foamy cytoplasm appeared to be degenerating, though no fragmented nuclei were found. About some of the larger venous sinuses there was a small number of lymphocytes.

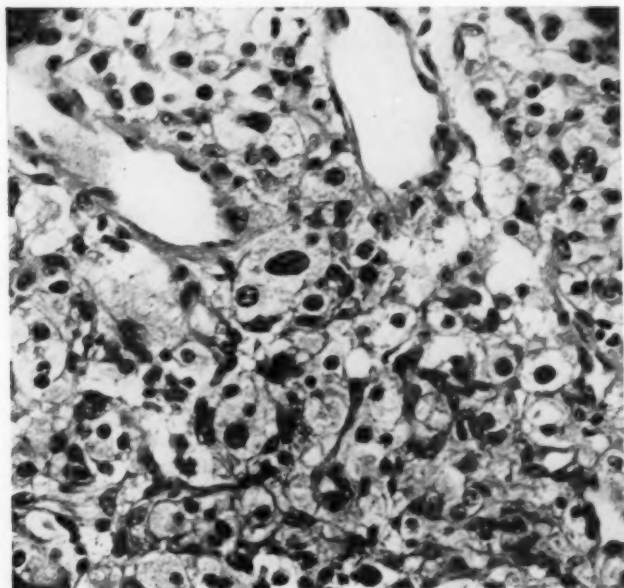


Fig. 3. Tumor of ganglion nodosum. There is marked variation in nuclei (Hematoxylin and Eosin x600).

DISCUSSION.

Tumors of the carotid body and related structures, including the glomus jugulare, the paraganglion tympanicum, the ganglion nodosum of the vagus, and the aortic bodies have recently been reviewed by LeCompte¹. These structures were once erroneously considered to be paraganglia, to be endocrine

glands secreting epinephrine, and to stain green with chrome salts. They have more recently been shown to be chemoreceptor organs sensitive to changes in the pH, carbon dioxide content and oxygen tension of the blood. They are not endocrine glands, nor are they paraganglia. The histogenesis of the structures is in dispute, some claiming that they are neuroectodermal; others, that they are mesodermal in origin. The term chemodectoma has been proposed for tumors of chemoreceptor organs.

The present case is the fifth tumor reported, which arose in the ganglion nodosum of the vagus nerve^{1,2}. The reported ages have been from 28 to 49 years, and all but one have been in women. In three of the five cases there were also tumors in other chemoreceptor organs. Although a familial incidence has been shown in carotid body tumors, the present case is the first in which a tumor of the ganglion nodosum occurred in a family, another member of which had a chemoreceptor tumor.

All of the five tumors were surgically excised. In one tumor it could be only incompletely surgically removed, and the patient was given X-ray therapy. He died five months later of poliomyelitis without symptoms referable to the tumor. Another patient died of bronchopneumonia and pressure of a recurrent tumor on the brain stem, 32 months after incomplete removal.

SUMMARY.

A carotid body-like tumor of the ganglion nodosum of the vagus nerve is reported, and four other similar tumors are discussed.

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A CONTRIBUTION TO THE PROBLEM OF THE BLUE EARDRUM: IDIOPATHIC HEMOTYMPANUM.

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"Blue eardrum" is a clinical symptom caused by the addition of the hue of blood to the tympanic membrane. The symptom is frequently seen in plain traumatic hemotympanum. In other cases it has been reported as a symptom of the jugular bulb protruding through a dehiscence into the middle ear, or of a hemangioma behind the eardrum; however, there remains a number of cases to which the name of idiopathic hemotympanum has been applied and which seem to represent a definite disease entity. Only 14 such cases have been reported in the literature although it is thought that many more have actually occurred. Recently Johnston⁶ has given a full review of this syndrome, which is characterized by the insidious and painless onset, the copious viscid brown fluid in the middle ear, and the tendency of this fluid to reaccumulate without spontaneous cure. The minimal symptoms are in contrast to the surprising degree of conductive hearing loss. In no instance have hematologic or systemic disorders been found to explain the bleeding tendency.

Considering the few cases reported it seems justified to report another case of idiopathic hemotympanum, especially as this case offers an unusual feature, which may possibly throw some light on this rather puzzling disease.

CASE REPORT.

The patient, a white female, was seven years old when first admitted to the Roskilde County Hospital Nov. 5, 1950. Her previous history was non-contributory. Fourteen days before her admission she suffered a skull trauma by falling from a chair. There was no apparent ill effects after this event, but one week later she developed a spontaneous otorrhea from the right ear, followed within 12 hours by a complete

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facial paralysis on the same side. On admission there was a copious, brownish, viscid discharge from the right ear, where the tympanic membrane was thought to be diffusely swollen, and a homolateral complete facial paralysis. The mastoid region was found to be normal, and X-ray examinations of the skull revealed no fracture. On penicillin therapy the ear became dry in ten days, when it became apparent that the drum was dark blue, and remained essentially so despite inflations by the Politzer and the catheter method. The facial paralysis improved slowly on treatments with electrical stimulation, massage, and vasodilating drugs, and upon her discharge Jan. 9, 1951, it had disappeared almost completely.

Three months later, after an acute upper respiratory infection, there was a recurrence of the right otorrhea, and the patient was readmitted to this department April 11, 1951, with copious, brownish, viscid discharge from the right ear, and a swollen, red tympanic membrane. On penicillin therapy the ear became dry in three days. On the fifth day the drum was almost normal, but on the eighth day it resumed the previous dark blue color. An audiogram made at this time revealed a 20 decibel conductive hearing loss in the low frequency range for the right ear.

The patient remained well until three years later, when, after a severe febrile upper respiratory infection she had earache on the right side, followed shortly by a copious brownish discharge from the ear, which was treated successfully with penicillin. Fourteen days later there was recurrence of the fever, which responded to treatment with a sulfa drug; however, the purulent discharge from the nose and deafness in the right ear persisted. After one month, April 8, 1954, she was readmitted to the hospital. Clinical examination revealed a slightly bulging, dark blue eardrum, and some mucopurulent secretions in both nasal cavities. Roentgenograms showed clouding of both maxillary sinuses, and by irrigation pus was recovered from the left side. The sinusitis was treated with sulfa drugs and repeated irrigations and abated within a week. As the drum remained unchanged, a generous myringotomy was performed, with removal of a large amount of brownish mucoid fluid. The drainage of this fluid persisted for two days, and under treatment with repeated inflations and chloromycetin there was a definite decrease of the intense blue color of the drum. Gradually, however, the fluid reaccumulated and the hearing remained severely impaired. Roentgenograms of the mastoids showed a normal left side, while there was a remarkable increase of the pneumatization on the right side, with clouding of the whole cell system but without evidence of destruction.

Considering the previous history it was now decided to perform a simple mastoidectomy, which was done on May 12, 1954. The mastoid was found to be most extensively pneumatized, with the cells being very large and the septa sclerotic. All the cells were filled with mucoid, brownish secretions, except in the apical and deep subantral regions, where the secretions consisted mostly of pure old blood. In this region also the mucous membrane was very thick, in some places resembling granulation tissue. Because of the previous palsy, special attention was paid to the facial canal. It was not thought advisable to open it because of the risk of a recurrence of the palsy, but the osseous covering was friable and in some places not very well marked. Bleeding was only slight, and no area was encountered resembling angioma, nor were there any signs of a previous fracture. On termination of the operation all cells and mucous membrane had been removed, and there remained a very large cavity. The postauricular incision was closed after insertion of a rubber tube into the antrum.

Histological examination of a tissue specimen from the subantral region showed small tissue fragments, consisting essentially of densely cellular,

proliferating tissue with very few fibrils, and only moderately vascularized. It contained numerous cholesterol crystals, some blood pigment, and a considerable number of foreign body giant cells. The intermediate osseous tissue appeared essentially normal. There was no inflammatory reaction, granulation tissue, or hemangioma. The general impression was that of an old hematoma in the process of organization and with poor absorption.

The postoperative course was uneventful, with no symptoms from the facial nerve. For three weeks there was some brownish discharge from the wound, which was purposely kept open with rubber tubing. Eighteen days following the operation inflations of the middle ear were started and continued for one month after the patient's discharge from the hospital June 12, 1954. During this time the drum retained a slight discoloration over the lower part. On the follow-up examinations in January and April, 1955, the eardrum appeared normal. Audiometry revealed an average hearing loss of 20 db., with no change after inflation of the middle ear.

DISCUSSION.

For lack of conclusive evidence of any definite pathology, the etiology of idiopathic hemotympanum still remains in the field of philosophy, mainly dominated by the analogy, which in certain respects exists to serous otitis media, and also by speculations as to how spontaneous hemorrhages may occur in the middle ear cleft, and the blood fail to become absorbed, in contrast to cases with traumatic hemotympanum. Consequently Johnston⁶ arrives at the conclusion that somewhere in the middle ear cleft of these patients there is an area of varicosity or even hemangioma, which fails to undergo complete tissue repair, and repeatedly breaks down to permit the escape of fresh blood.

In the above reported case there is the additional feature of a facial nerve paralysis, which appeared at the same time as the ear symptoms. This paralysis might be due to an otitis media, although it did not develop until after the onset of otorrhea; furthermore, the paralysis was complete and persisted longer than is the case when caused by otitis media. One week prior to her symptoms she suffered a skull trauma. The history of a trauma was elicited under direct questioning and it might have been very slight; however, the elapse of one week seems to exclude any direct relationship between the paralysis and this trauma. It seems most natural to explain the paralysis as being a Bell's palsy. How the otorrhea fits in with this is not quite clear; however, on

admission the secretions were brownish and viscid and not purulent, as would be expected in an otitis media of long standing. Unfortunately, the secretions were not cultured and it is not known whether they were sterile.

By now it is generally accepted that most cases of so-called Bell's palsy are due to some kind of vascular obstruction around the stylomastoid foramen, causing ischemic changes in the facial nerve, with swelling of the nerve within the bony canal and secondary compression (Audibert et al.¹, Cawthorne², Sullivan¹⁰, Kettel⁷, Hilger³). As a further result of the ischemia, in severe cases, exudation, or even a hemorrhage, into the mastoid cells may exist, together with bony necrosis of the walls of the Fallopian canal (Kettel⁷, Hall⁴, Flodgren³). This necrosis is of a purely degenerative type and never inflammatory. The present case might fit into this description, except that the secretions were so abundant that some kind of low grade infection must be suspected. The partial response to antibiotic therapy, and also the repeated exacerbations, in conjunction with upper respiratory infections, support this view. A necrotic area in the mastoid must presumably be very susceptible to infections by way of the middle ear or the blood stream. The findings at operation helped to localize the diseased area definitely to the region around the lower portion of the facial canal. Histological examination revealed no bony necrosis; however, much time had elapsed since the first acute episode, and such necrosis was hardly to be expected. As mentioned, we dared not expose the nerve for fear of recurrence of the paralysis.

In the latest report of a case of idiopathic hemotympanum Simonton⁹ mentions that his patient suffered from homolateral episodes of hemifacial spasms as well. During the radical mastoidectomy he found that the bony wall covering the horizontal portion of the Fallopian canal was absent, and the VIIth nerve in this area was enlarged approximately to four times its normal diameter. It might be stated, that at least some cases of hemifacial spasms are brought about by the same changes in the vascular supply around the stylomastoid foramen, as in other instances would produce a Bell's

palsy; in fact, some cases do appear following Bell's palsy (Williams, Lambert and Woltman¹¹, Kettel⁸).

Thus we have two cases of idiopathic hemotympanum with two different concomitant diseases, both pointing towards a common etiology. During the future treatment of such patients it might be well to keep in mind the possibility that a variety of disturbed vascular circulation in the area supplied by the stylomastoid artery may exist.

SUMMARY.

A report is given of a patient with concomitant idiopathic hemotympanum and Bell's palsy, cured by simple mastoidectomy. It is suggested that some cases of idiopathic hemotympanum are due to complete or partial vascular obstruction around the stylomastoid foramen, affecting the nutrition of the facial canal and the surrounding mastoid cells.

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Aabrinken 205.

THE RESPONSIBILITY OF THE DOCTOR TO THE HARD-OF-HEARING PATIENT.

FRANK S. FORMAN, M.D.,*

and

ARMIN E. GRABER, M.A.,

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The hard-of-hearing patient for whom remedial therapy has failed, or for whom it was deemed useless, represents a perplexing problem for the community and a challenging problem for the physician. That these people need effective help is immediately apparent when we consider their handicap. The poignant problem of the deaf has long been recognized by our communities, and has been met by our State educational programs and schools for deaf children.

The acquired deafness in the adult, many of whom are elderly, has not been given more than cursory attention. These deaf do present a social, moral, and almost ethical problem that the doctor of medicine cannot find to be unsurmountable. These people can no longer hold their responsible jobs because of their inability to understand directions sufficiently to make their work reliable, or because the frequent necessity of repetition creates an atmosphere of exasperation. Employers will not and cannot continue to accept the responsibility for the deafened employees' errors or continue to accept the exasperation, with the result that these handicapped persons are discharged from employment and are cast out into an unsympathizing community. These suffering people have been neglected by almost everyone, the doctor in particular.

The deaf try to help themselves by securing hearing aids and only a small fraction are fully satisfied with the aids. Even those who do not become discouraged with their first aid and who have the means and the persistence not to give

* From the Hearing Institute of the Colorado Springs Medical Center.
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up hope, seldom find anything satisfactory except the cheerful promises of the hearing aid salesperson.

The fact that we do not try to help these people is not altogether our own fault but is the result of various cumulative factors which have inevitably developed in the evolution of the hearing aid and the hearing aid industry. To say that we alone are at fault is flagrantly incorrect; it is time for us to review our positions, correct our errors, and individually help the deaf.

In order to understand our position, we must consider what a hearing aid is and what we can expect from it. A hearing aid is nothing more or less than a miniature sound amplifying system, a small individual public address system. For a hearing aid to do more than one could expect from a public address system would be inconceivable. We theorize that if a deafened person can hear a radio he should be able to hear with its miniature counterpart, a hearing aid. Unfortunately, only a few hearing aids today remotely approach the reproduction of communicable sound by large amplifying systems.

This lack of faithful reproduction in hearing aids represents one of the reasons of propitious importance why we physicians have been passive in recommending hearing aids. A doctor takes legitimate pride in his work, and is constantly trying to achieve something better for his patients. From practical experience in some instances, or from hearsay evidence with his hard-of-hearing patient, the doctor hesitates to recommend even the very best available hearing aid if it does not meet his personal specifications of being more than adaptably useful; furthermore, a doctor is a man of science; he cannot understand why, if sound can be reproduced faithfully in his everyday life in radio, TV., etc., it is not possible to reproduce the same thing in a hearing aid. He will not accept responsibilities for recommending something he believes is not good.

Another reason for our apathy in prescribing hearing aids is the admission of defeat of our sincere therapeutic efforts. When a hearing aid is advised it should not be construed that

we have failed medically. We do not hesitate to advise glasses or an artificial limb. In these we know our capabilities and limitations, and do not expect to do the impossible. To admit defeat in deafness does not preclude any concept of further study or trial of methods for restoration of hearing. No, we should not despond, but we should be practical enough to weigh judiciously the evidence at hand, to evaluate the usually remote possibility of restoration of the patient's hearing and to discuss his problem with him.

The evolution of the hearing aid industry very clearly shows another reason for our present status as physicians in the fitting of hearing aids. There is a remarkable parallelism between optic and acoustic appliances. It was not until eye glasses became perfected, and it was not until the basic physics involving refractory errors were known that physicians prescribed glasses. At first the itinerant fitter who had magnifying lenses was the only person who attempted optical correction. It was later when the grinding of lenses became perfected that optometry became a recognized business, based on trial and error correction. Only after the refractive errors could be measured and correlated to the pathology present did doctors accept responsibility for prescribing glasses.

In the hearing aid industry the ear trumpet could hardly interest the scientific man. With the development of electrical acoustic devices better amplification could be produced. At the present time these hearing aids are vastly superior to the initial ones, and at least one make can be compared to the efficiency of glasses. Commercialism has so entered the situation today that most aids have progressed in some respects no further than the lenses sold by itinerant eye-glass peddlers.

It is likewise appalling that the hearing aid industry has not taken it upon itself to educate the doctor more often as to the merits of its product. The pharmaceutical companies spend time and money telling us of their products directly through detail men who inform us of their particular products as well as advancements in general. In a sense we feel responsible for some of these pharmaceutical advance-

ments, because we have more or less demanded better medicines through the detail men, and we have worked together.

The hearing aid industry has not done anything comparable to this. Perhaps if we had closer contact with them we could understand their problems and they could understand what we require. This is not necessarily one sided, for it is our responsibility to understand hearing aids; however, the information available to us from many hearing aid representatives is so meager and so unscientific that it is often worthless. Steps are, however, being made in the direction of the physician-hearing aid industry cooperation. This will make possible mutual understanding, cooperation, and help.

Yet another reason for our not being interested in the fitting of the hearing aid is the lack of our ability to predict much from our audiometric testing. Pure tone audiometry, which is the routine method of office testing, is lamentably inadequate. Pure tone audiometric testing is based upon threshold response at different frequencies. This does not test hearing acuity, and represents only a comparable test of the patient's ability to see the smallest amount of lights of different colors. No ophthalmologist would ever prescribe glasses based on these measurements. Indeed he could not, because he is perfunctorily interested in color perception or threshold vision for correcting optic defects.

Even while there are certain correlations between pure tone audiometry and speech, this paradox in threshold testing persists. Our present routine methods of testing have little if any place in hearing aid fittings. Hearing aids must be and are fitted in reality, without regard to the pure tone threshold audiogram except for the rough acoustic gain required and the flat and high tone emphasis. This is not good. Further investigation and further application of our present knowledge of methods, other than pure tone threshold audiometry, will enable us to predict the applicability of hearing aids for our deafened patients.

From these few factors alone it is easily understood why we have helped the hard-of-hearing adult so little. What can we do about it? The patient comes to us for help, so

let us help him in the only direct way we can. We must fit hearing aids. Let us select the best aid for each individual patient. Let us make an impression for the ear piece and see that it fits properly. Let us educate the patient to use his aid in noisy environments by the simple application of simulated noise environments in our office with phonograph records; then let us aid in training them to select speech from the non-essential background noise and to understand this speech. Let us give the psychological boost to the patient in order that he does not become discouraged. Finally, let us help the hearing aid industry by finding the best aid and fostering the acceptance of efficient, faithfully reproducing amplifying systems which are forthcoming with development of the electronic industry. Only by doing these things can we fulfill our obligations to our hard-of-hearing patients and to our community.

POST GRADUATE COURSES AT TEMPLE UNIVERSITY

Postgraduate Course in Bronchoesophagology, February 11th - 22nd, 1957 — May 20th - 31st, 1957 — September 9th - 20th, 1957.

Postgraduate Course in Laryngology and Laryngeal Surgery, March 11th - 22nd, 1957 — November 4th - 15th, 1957.

These courses are all to be given in the Department of Laryngology and Broncho-Esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is \$250.00.

Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia, 40, Penna.

OSTEOMA OF THE EXTERNAL AUDITORY CANAL, CASE REPORT.*

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and
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Beverly Hills, Calif.

The rarity of osteoma of the external auditory canal large enough to cause symptoms has been established¹. O. R. Kline and R. C. Pearce were able to find reports of only 36 cases, and added an additional case. Although some cases have been treated by excision through the external auditory canal, by far the majority have required some type of mastoidectomy. Following is the report of a case treated by excision through the external auditory canal. Preoperative X-ray studies in this case were of considerable help in delineating the extent of the tumor in relation to the middle ear.

The patient was a 44-year-old housewife who had known of a mass in the left ear as long as she could remember. For at least ten years she had had periods of marked hearing loss whenever she got water in the ear. These periods would last from one to two months and would sometimes be accompanied by pain and a thin discharge. In between attacks she noted no loss of hearing.

Examination showed a bony hard tumor occluding the left external auditory canal. The surface was smooth and covered with skin, with its lateral surface deep to the edge of the auricular cartilage. After cleaning off all epithelial debris from the edge of the tumor a very narrow slit was found between the tumor and the canal inferiorly. X-ray in the submento-vertex position showed an indistinct mass in the left external auditory canal with an air space medial to it. It was not possible to get a needle through the slit below the tumor because of pain; however, a tiny flexible polyethylene tube (approximately the size of a No. 23 needle) could be passed beyond the tumor for a depth of 2 cm. Through this tube Diodrast was injected, and X-ray in the Towne position (see Fig. 1) clearly outlined the medial aspect of the tumor and the tympanic membrane. It was then felt that removal through the external auditory canal could be safely accomplished.

At operation on February 1, 1956, the findings bore out the X-ray studies. Using a small dental burr with the contra-angle attachment a small opening was made in the inferior area of the tumor, which was known to be thin. This opening was gradually enlarged until it was

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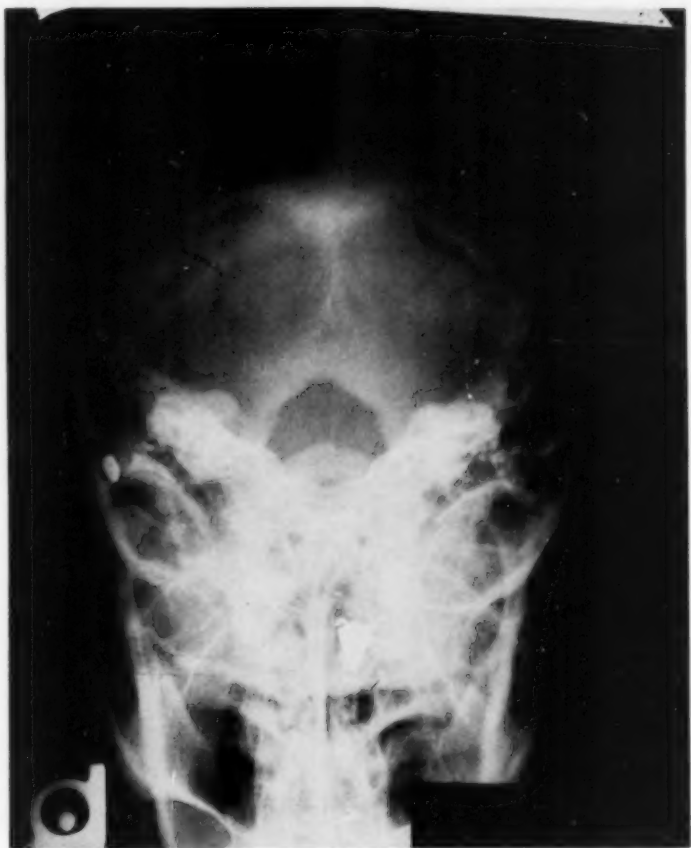


Fig. 1. Roentgenogram of mastoids in Towne position showing Diodrast in the air space between the tumor and the tympanic membrane. The circular opacity is a lead disc lying against the lateral surface of the tumor.

possible to keep the tympanic membrane in view. After the canal had been enlarged to about 6 mm. the anterior rim of the remaining tumor mass could be identified separate from the antero-superior margin of the bony canal wall. This rim was engaged with a curette and separated rather easily *en masse* from the canal. The surface of the tumor was smooth and covered with connective tissue. The canal was of normal contour and the tympanic membrane was intact. The tumor measured

8 mm. in its largest diameter and was semilunar in shape (see Fig. 2). Microscopic report was that of a typical osteoma.

Two weeks postoperative the canal was healed and of normal appearance. The audiogram was normal showing no change from the pre-operative level.

COMMENT.

Because we were able to get Diodrast between the tumor and the tympanic membrane, it was possible to determine that



Fig. 2. Tumor after removal.

operation could be safely done through the external auditory canal. Even if a contrast medium could not be introduced it is possible that careful X-ray studies, including lamina-grams, could give the same information and sometimes avoid mastoidectomy.

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436 No. Roxbury Drive.

THE RESEARCH STUDY CLUB OF LOS ANGELES

Announces its Twenty-Sixth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 14 through January 25, 1957.

For "Ear-Nose-Throat," the main Guest Lecturers will include Dr. Louis H. Clerf, Dr. George W. Shambaugh, Jr., and Dr. Maurice H. Cottle.

Mrs. Spencer Tracy, Founder and Director of the John Tracy Clinic of Los Angeles for Deaf and Hard-of-Hearing Children and Their Parents, will speak to the "Ear-Nose-Throat" group the opening day of the Convention on "The Role of Parents in the Education of a Deaf Child."

Dr. H. Russell Fisher, a pathologist of note, will bring us up-to-date in his field.

Instruction Courses in Otolaryngology will be provided by three main speakers, Dr. Clerf, Dr. Shambaugh and Dr. Cottle, as well as by Dr. Aram Glorig, Jr., and Douglas E. Wheeler, Ph. D., both of Los Angeles and authorities in Audiometry.

The main Guest Lecturers for "Eye" program will be Dr. John M. McLean and Dr. Graham Clark, both of New York City; and Dr. Samuel J. Kimura of Berkeley, Calif.

Instruction Courses in Ophthalmology will be conducted by Guest Speaker, Dr. Kimura; Dr. Julian N. Dow, Dr. Robert V. Shearer, Dr. Alfred R. Robbins, and a symposium composed of Dr. Clarence H. Albaugh, Dr. John P. Lordon, and Mr. Russell L. Stimson—all of Los Angeles.

The first week will be devoted to the "Ear-Nose-Throat," from Monday to Friday, January 14 through January 18. The "Eye" week will cover Monday through Friday, January 21 through January 25. Those who confine their work to only one of these specialties may complete either subject in one week, or remain for the full two weeks.

As a period of relaxation, Wednesday afternoons will be

free for sight-seeing, shopping, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

Motion pictures of "Ear-Nose-Throat" subjects will be shown during the first week—Monday, Tuesday, Thursday and Friday, 3:00 to 4:00 p.m.

"Eye" subjects will be presented on Wednesday afternoon of the second week for those who prefer to take advantage of this opportunity to view films dealing with subjects in ophthalmology.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting in San Francisco between the two weeks of the Convention on Saturday afternoon, January 19, and Sunday morning, January 20. This will permit members to attend the Triologic meeting and return to Los Angeles for the "Eye" week. Programs of the San Francisco meeting will be available at the registration desk at the Elks Club.

On Monday evening, January 21, the annual banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of the "Eye" week. This will take place at 7:00 p. m. in the El Venado Room of the Elks Club.

The Women's Entertainment Committee will arrange for various events of interest to the wives of visiting members during the Convention. This Committee will appreciate information as to whether your wife will attend with you, which will give the Committee an estimate as to the number of wives for whom to plan entertainment. Details of all such events will be printed and available at the registration desk.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. R. B. Philbin, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail. The Elks Club is the locale for the entire daily program.

All applicants must be members in good standing, of the American Medical Association, *in order to become eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is \$100.00 and includes the cost of all luncheons. These dues are an income-tax-deductible item, as they represent an Annual Membership Fee. Make your check payable to "Mid-Winter Clinical Convention," and mail to Norman Jesberg, M.D., Treasurer, 500 South Lucas Avenue, Los Angeles 17.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

The Officers and the Organizing Committee of the Sixth International Congress of Otolaryngology cordially invite you to attend the Congress which will be held in Washington, D. C., U. S. A., May 5 through 10, 1957.

REGISTRATION AND OPENING.

Any qualified physician may become a Member of the Congress by paying a registration fee of \$25.00, U. S. A. Non-medical personnel may register as Associate Members for a fee of \$10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

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Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

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Meeting: Statler Hotel, Washington, D. C., May, 1957.

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Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

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Meeting: Palmer House, Chicago, Ill., Oct. 13-19, 1957.

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Meeting: Palmer House, Chicago, Ill., October 6-12, 1957.

THE AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chesson, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois,
October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1957.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

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Meeting: Palmer House, Chicago, Ill., October, 1957.

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Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14,
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Meeting: Palmer House, Chicago, Ill., October, 1957.

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PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Phila-
delphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and
Broncho-Esophagology.
Time and Place: Brazil, 1958.

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Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

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Meetings are held the second Tuesday of September, November, January,
March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

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**WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY
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Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18,
1957.

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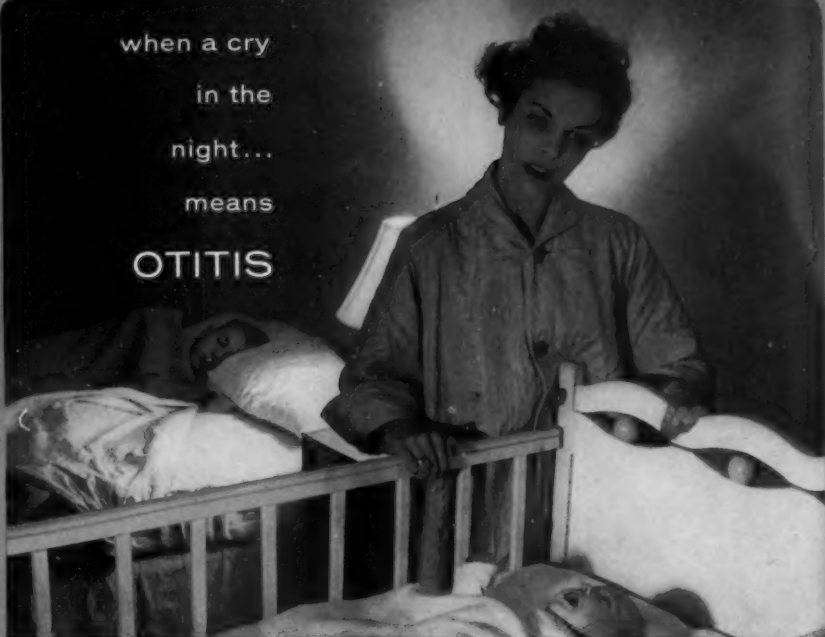
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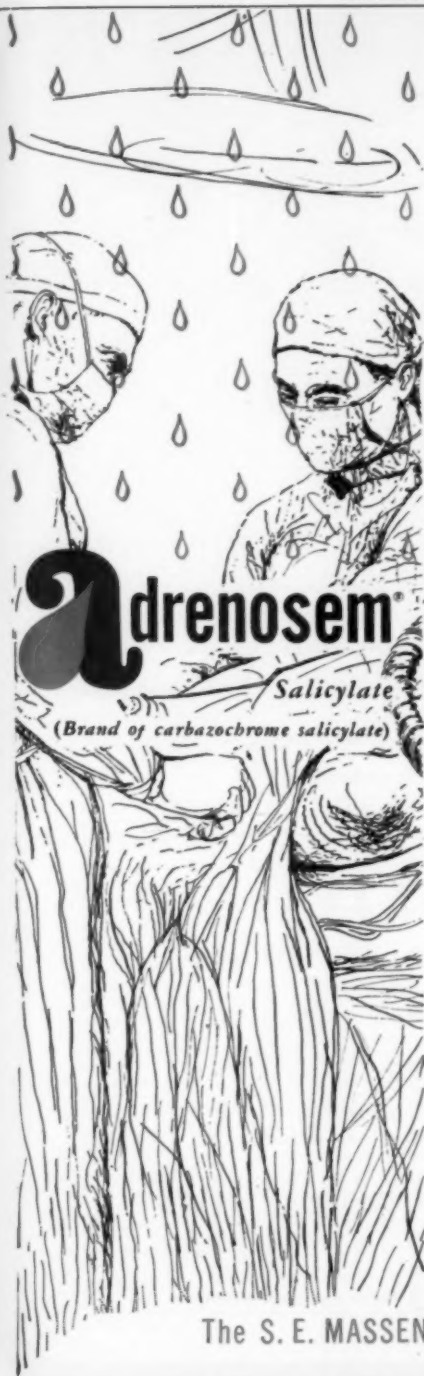
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1. Lazar, A. M., and Goldin, M.: Eye, Ear, Nose, & Throat Monthly 33:351, 1954. 2. Lazar, A. M., and Goldin, M.: Antibiotics Annual 1954/5, p. 468.

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1. Lazar, A. M., and Goldin, M.: Eye, Ear, Nose and Throat Monthly 32:512, 1953.

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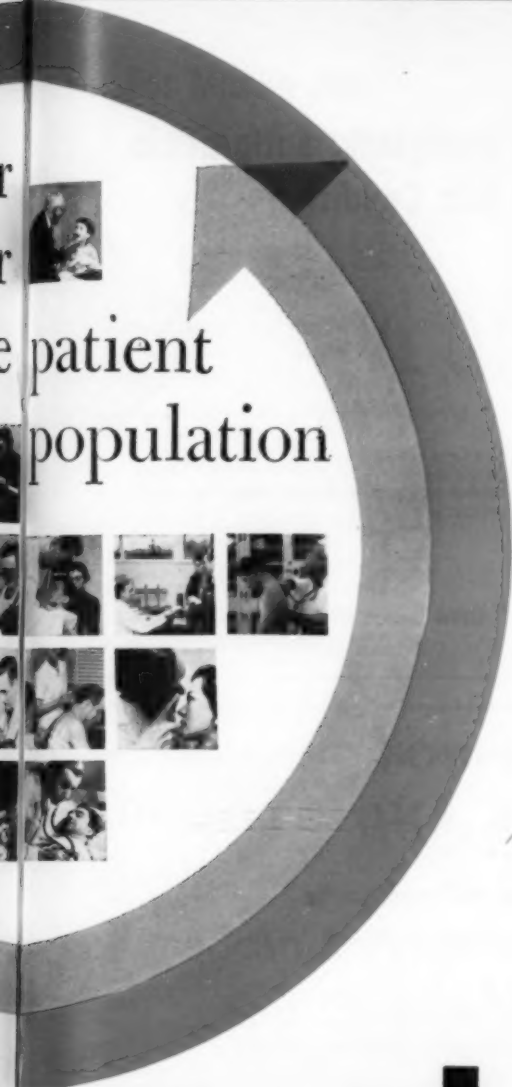
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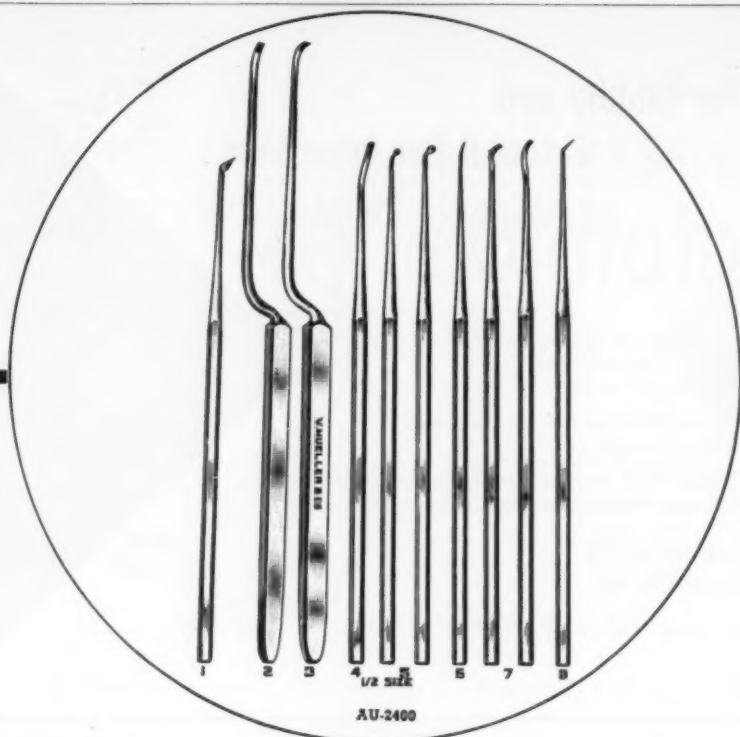
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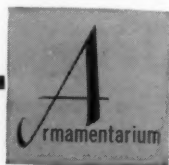
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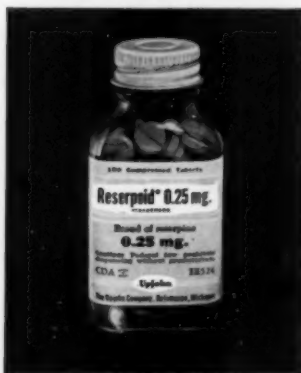
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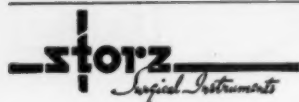
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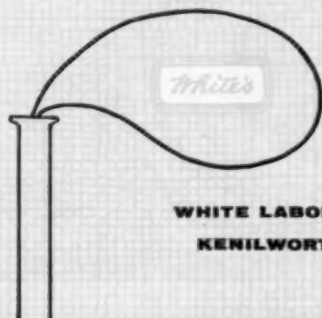
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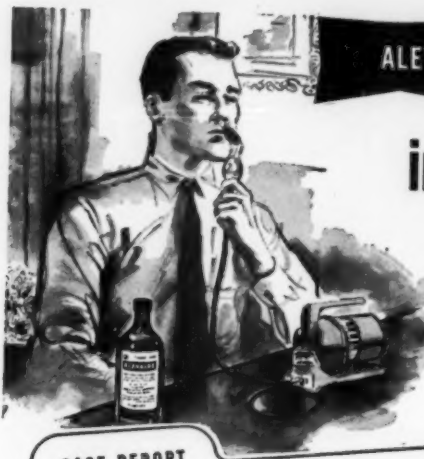
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*Miller, J.B., et al.: Ann. Allergy, 12:611, Sept.-Oct., 1954.

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